

VETERINARY DENTISTRY FOR THE GENERAL PRACTITIONER AND VETERINARY TECHNICIAN

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The goal of this course is to cover give a broad overview of veterinary dentistry in a fashion that enables the veterinarian and the technician to make good decisions about how to go forward with the dental practices and how to take their dental practice to the any chosen next level.

The majority of patients brought in to see veterinarians have some form of dental pathology. Dogs and cats do not show dental pain. To realize this fact is of paramount importance. Otherwise our patients might walk around day to day with dental pain and be suffering in silence. Appropriate dental treatment can not only prolong the lives of our patients but it can dramatically increase their quality of life. Over and over, patients come to me for their recheck exam after a dental procedure feeling so much better that clients can feel guilty they did not do the procedure sooner. Inappropriate dental care is worse than no care at all as it often increases the patients pain level, involves an anesthesia that yielded fruitless or negative results, and gives the client a false sense that the animals needs have been addressed.

This course will cover basic dental terminology to “make life easier” and charting more expedient in the dental room. Dental radiography technique will be taught in a fashion that will increase speed and accuracy. The goal for your practice is to be able to perform the majority of full mouth radiography sets in under 10 minutes. This is such a win-win for the patient and the practitioner. Interpretation of radiographs, some commonly seen oral pathology as well as some less commonly seen oral pathology will be covered.

For dental care to be useful it needs to involve at least 15 components of a COHAT (Comprehensive Oral Health Assessment and Treatment). Though this can sound overwhelming at first, many of the items (and for some all of them) are already instituted in practices routinely. Component (1): Obtain a thorough patient history and signalment. Ask the client about home care and chewing habits. Component (2): Do a thorough general physical and awake oral exam. Component (3): Obtain preanesthetic diagnostics based on age and health status of the patient. Component (4): Make a preanesthetic plan. For instance, patients with chronic pain should be given an IV analgesic profusion prior to the procedure. Component (5): Anesthetize the patient with induction, endotracheal tube placement and gas anesthesia maintenance. Component (6): Use a comprehensive 6 parameter monitoring machine that monitors O2 saturation, ET CO2, EKG, respiration, blood pressure and temperature. Component (7): Use a external warming system like a Bair Hugger or a warm water circulating blanket or both. Component (8): Constantly monitor with three team members, doctor, dental technician and anesthesia technician present. Component (9): Visually assess the entire mouth, do dental radiography of every tooth and surrounding tissue, do periodontal probing and chart your findings. Studies have proven that dental radiography will show something you did not discover on oral exam more than 90% of the time and most of the time what is found requires treatment. Component (10): Complete scaling above and below the gum line and then polishing is done. This cannot be performed adequately or humanely without anesthesia. Component (11): Formulate a treatment plan. Now the owner is contacted by phone with the updated plan to get permission to do the needed procedures.

Component (12): Oral nerve blocks are administered. This allows for a lighter and safer plane of anesthesia. Patients wake up faster and remain pain free for up to 6-10 hours postoperatively. Component (13): Now its time to implement the treatment. Seldom is treatment limited to a dental prophylaxis. By far the most frequent procedure performed is a periodontal flap based surgery allowing disease tissue and bone to be removed and sutured over extraction and open root planing sites. Extraction of teeth without gum flaps dose little to nothing to benefit the patient if all the disease surrounding the tooth is left. This is definitely surgery and should only be done by a veterinarian well trained in oral surgery and radiographic interpretation. Other procedures may be recommended to save teeth such as guided tissue regeneration, root canal therapy, or bonded restoration. Component (14): Upon completion of the procedure the oral cavity is flushed, the pet wakes up quickly and the owner is called to pick up their pet. Component (15): The final step is discharge of the patient during which medications, instructions for home care, contact phone numbers, and recheck appointments are thoroughly discussed.

When talking about teeth, a few basic dental terms can be simple and easy to learn and and serve you well. It is important to realize that the terms rostral and caudal loose their meaning when speaking of tooth surfaces because teeth present in an arcade. The **mesial** surface of the tooth is the part of the tooth toward to the midline of the arcade. The **distal** surface is away from midline. **Palatal** surface is toward the hard palate on the upper arcade while the **lingual** surface faces the tongue on the lower arcade. The **buccal** surface of a tooth faces the cheek and the **labial** surface faces the lips. The **occlusal** surface is the part of the tooth that occludes with other teeth. **Apical** is toward the root and **coronal** is toward the crown.

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FELINE DEGENERATIVE JOINT DISEASE

Ilona Rodan, DVM, DABVP, Feline

Introduction

Degenerative joint disease (DJD) is a common but under-diagnosed condition in cats. Although the frequent occurrence of feline DJD was only recognized in the last 10-15 years, there is a plethora of journal articles that address both the challenges and measures taken to diagnose and treat this condition.

There are multiple reasons for the difficulty to diagnose DJD in cats. Cat owners think their cat is just slowing down due to aging. The placebo effect is very high in studies with owners attempting to assess efficacy of treatment and adverse events.¹ Veterinarians have more experience diagnosing arthritis in dogs, but cats with DJD rarely demonstrate the more obvious signs recognized in dogs. Gait analysis in the practice is especially challenging in feline patients.

Even when diagnosed, there is still a lack of or inadequate treatment of feline DJD due to concerns about adverse events from drugs and owner difficulty to administer medication. DJD impacts the cat's quality of life and the relationship that owners have with their cats.

Incidence of DJD

Degenerative joint disease is a common cause of chronic pain in cats.^{2,3,4,5} In one random study of cats in different age groups, 91% of 100 cats had radiographic evidence of DJD, occurring as early as 6 months of age, and with equal frequency in all age groups.⁵ Signs appear to worsen with age.⁵

Concurrent conditions occur frequently, and were found in 44% of cats affected with DJD in one study.⁶ Although concurrent disease is common in older cats, chronic kidney disease and DJD occur concurrently in all age groups.⁷

Is it DJD or arthritis in cats?

The following terms - arthritis, osteoarthritis, or DJD – are found in different journal articles addressing this feline condition. However, arthritis is defined as inflammation of the joint, whereas DJD consists of both inflammatory and non-inflammatory disease processes. The latter is what occurs in cats, leading to the degeneration or destruction of synovial (appendicular) or cartilaginous (intervertebral disc) joints.² It is osteo-productive leading to the development of osteophytes.

Where does DJD occur?

Feline DJD occurs in both the spine and the appendages. Spinal or axial DJD is more frequently found between thoracic vertebrae T7-T10, but the lumbar vertebrae are affected more severely. Axial DJD increases with age.⁶

The more commonly affected appendicular joints are the hips, elbows, knees, and hocks. As opposed to axial DJD, appendicular occurs equally through the ages.⁶

The challenge to diagnose

Feline DJD is difficult to detect because of the cat's tendency to hide pain as a protective mechanism. Cat owners think their cats are slowing down or "just getting old". Additionally, as opposed to the dog, most cats with DJD don't limp because the disease is bilaterally impacting the same joints.⁸

Treatment trials have been and continue to be done to identify treatments for feline DJD. However, the placebo effect is huge with owners noting improvement in both placebo and treatment groups.^{1,9} In one study, differentiating between placebo and treated cats was only recognized once treatment was withdrawn.¹ Also of note is that the placebo group often had a high rate of adverse events reported.^{10,11}

Although many cats have radiographic evidence of DJD, radiographic signs do not equate with pain. Additionally, cats that have early DJD without obvious radiographic changes consistent with DJD can be painful. This makes owner input even more important.

Changes in behavior are the most common signs of DJD, but they also occur with other physical pain, either acute or chronic, non-painful illness, as well as with emotional pain, such as stress. Additionally, waxing and waning of clinical signs is a feature of DJD.¹

A recent study indicated that joint palpation failed to differentiate between cats with DJD and those without, and that perhaps gait and body posture are most reliable to diagnose, but more information would be helpful.¹²

Recognizing pain through behavior changes

The signs of DJD are often subtle changes in behavior. These signs are so subtle that they frequently are unrecognized both by owners and veterinary professionals. The signs can be either changes in normal behavior(s) or the start of a new, but abnormal behavior for an individual cat, which can include behavior problems such as house soiling or aggression (see Table 1).^{2,3,4,6,10,13} A cat may present with one or multiple changes in behaviors.

Since changes in behavior are the most common signs of pain, the client is an important member of their cat's health care team because of their familiarity with their cat's behaviors and the ability to detect the earliest changes to those behaviors.^{2,3} Owner education is critical for them to recognize that even subtle changes are significant and to contact the veterinary practice if they notice deviations from their cat's normal behavior(s). However, signs of DJD are frequently only appreciated during the veterinary visit.

Diagnosing DJD

The history: Owner input is critical

Studies indicate that clients often recognize the pain of DJD in their own pets more accurately than veterinarians because they know their cat's normal behaviors and often recognize changes to the behaviors more readily.^{2,3,6,10,14} Unfortunately, clients frequently think the changes are associated with "old age" rather than pain. Owner involvement is also important to recognize response to treatment of pain.^{4,9} Interestingly, a study demonstrated that cat owners placed more

importance on non-physical outcomes (60%) such as grooming and comfort during resting, in contrast to the hypothesis that physical activity (mobility) would be more significant to owners.¹⁵

Changes in jumping, going up and down stairs, and hesitation to jump or climb are signs that owners should watch for in addition to all other behavior changes noted in Table 1. Letting owners know that purring is often used to comfort self, and can occur in painful cats.

History should include open-ended questions about changes in behavior.¹⁶ For example: “What changes have you noted in “Fluffy’s” behavior since the last visit?” A good follow-up question is: “What else?” If the owner has not mentioned changes in the cat’s gait, jumping, or step climbing ability, ask if changes have been noted in any of these or show a video or image that indicates possible signs. Sometimes, the signs of DJD are so subtle that they may appear as a hesitation to jump up to or down from a favorite spot, or moving more slowly going up and down steps. House soiling can also be seen with DJD, either because the cat cannot jump over the high sides of the box or because they cannot climb the steps to the basement, the preferred litter box location for many owners.

Examination from a distance

While observing the cat prior to handling it, assess for stiffness and muscle atrophy over back and limbs. If the cat chooses to stay in the carrier, it is best to assess the gait at the end of the appointment, often following both examination and diagnostic testing. Cats should not be walked in a hallway as one would a dog due to fear, probable freezing or fleeing, and possible fear-associated aggression if the fleeing cat is chased. The easiest method to detect gait in the practice is to place the cat on the floor on the opposite end of the room from the carrier because most cats will immediately head towards the carrier, providing the veterinarian the opportunity to assess the gait. If there is not enough space in the exam room or if the cat slinks while walking at the hospital, ask owners to use readily available smart phones or other equipment to make video clips which can be submitted electronically for our review and to link to electronic records. Specifically request short videos of a cat preparing to jumping, climbing up and down stairs, and walking upon awakening.

Comparison with previous examinations can be very helpful. In addition to medical records, many hospitals have the capability to add patient pictures to the veterinary software. Use of this technology provides the opportunity to monitor changes such as the previously well-fleshed cat that has become muscle wasted either due to lack of usage with DJD or another underlying problem.

Hands-on examination

Patient handling should be done to prevent pain. Scruffing and stretching cats can exacerbate the pain of DJD. More information is provided in the next lecture.

A painful cat may be tense and resist examination in an attempt to protect self. Some cats that become aggressive with handling are painful cats. Gentle handling and providing analgesia will facilitate the exam and keep the patient as comfortable as possible.

Letting the cat remain in its preferred location and position, and tailor the order of examination, postponing the potentially painful areas until the end. Palpation of back and limbs should be

performed to identify painful axial and appendicular DJD respectively. Spinal pain is most commonly located over the lumbar and lumbosacral regions. Palpation of thickening of the elbow or knee joints is not uncommon with DJD of these joints. Other signs are crepitus, effusion within the joint capsule, and decreased range of motion.

Watching the gait following palpation is also helpful and can be done as the cat goes back to the carrier.

Treatment of feline DJD

The goal of multi-modal treatment is to target multiple sites along the pain pathways, and to potentially reduce doses of each drug to reduce the potential for adverse effects.¹⁷ Also, DJD treatment includes the need for both medical and environmental modifications to allow the cat to perform its normal behaviors and maintain comfort.

Pharmacologic Treatment

NSAIDs are the mainstay of pharmacologic treatment for DJD in cats as well as other species.^{4,18,19} NSAID's that are prescribed for cats should be used. These include meloxicam (Metacam) and robenacoxib (Onsior), but neither is approved for long-term use within the United States. Both however have been used in long-term studies^{1,10,11,13,19}, and meloxicam is approved for long-term in Canada, Europe, and several other countries. If meloxicam or robenacoxib are used long-term in the US, it is recommended that owners sign a waiver. Dosing should be by lean body weight. Owners should be warned to stop medication and call the veterinary practice if the cat is not eating, is vomiting, or any other changes. The patient should be reassessed for comfort as well as for diagnostic monitoring. The author does taper meloxicam to every other or every third day when possible.

Although veterinarians are often concerned about NSAID use in cats with concurrent chronic kidney disease (CKD), some studies have indicated safety at lower doses in cats with stable stage 1 or 2 CKD. One study indicated safety with 0.01–0.03 mg/kg *q* 24 hr).¹⁹

Medications that can be used in conjunction include gabapentin, Adequan, and mobility or joint diets.

Treatment efficacy

All cats should be rechecked to further assess for comfort and prevention of side effects. Many cats appear to do well with NSAID treatment in studies that are not double blinded. However, placebo effect is strong. Successful treatment has been measured during studies by activity monitors,¹³ and withdrawal of an NSAID, since differentiation did not occur in the placebo vs. treatment group in one study.¹

Non-pharmacological Treatment

The most important non-pharmacologic approach to treatment of feline DJD is modification of the home environment to allow easy access to favored places. Pet steps or ramps can provide easy access to preferred resting area for cats with DJD. Providing food, water, and litter in easily accessible areas where there is no competition for these resources improves feline welfare.

Acupuncture and weight optimization are excellent non-pharmacological treatments that can be used as a component of multi-modal therapy.

Table 1. Behavioral Signs of DJD in cats

Changes in the normal behavior of that individual cat

- Appetite
 - Decline
- Sleep/rest
 - Increase sleep or restlessness
- Grooming
 - Matting due to decreased grooming or overgrooming of the painful area
- Play
 - Decreased
- Toileting behavior
 - Difficulty getting into litter box
 - Change in position in box or toileting next to box
 - Constipation
- Activity
 - “Slowing down” or “getting old” – most common signs noticed by owners
 - Jumping and height of jump
 - Going up and down stairs
- Mobility
 - Stiff gait, may occur only when rises
 - Lameness – not common
- Disposition or attitude
 - Irritable: “Grouchy” or “grumpy”
 - Clingy
- Interactions with people or other animals
 - Withdrawn or avoid others
 - Attention-seeking
 - Irritable to aggressive with handling
- Body posture
 - Hunched

- Stiff
- Not curled up normally when sleeping
- Neck stretched out and head lowered
- Facial expression – fixed gaze, dilated pupils, squinted eyes if acute pain (flare-up)
- Vocalization
 - More or less vocal
 - Purring can occur even if painful

Behavior problems

- Inappropriate urination
- Inappropriate defecation
- Cat-to-human aggression
- Inter-cat aggression

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RECOGNIZING AND PREVENTING PAIN IN CATS

Ilona Rodan, DVM, Dipl. ABVP, Feline

Introduction

Pain management is essential to patient welfare, successful case outcomes, and client satisfaction.¹ Unfortunately, a lack of feline pain recognition leads to many cats not receiving analgesia or sufficient analgesia to adequately control the pain. A team approach that includes clients and all team members facilitates recognition and prevention of pain.

Recognizing pain through behavior

The client and pain recognition

Changes in an individual cat's behavior are the best method to identify pain in the cat.¹ Because the owner knows their cat and its normal behaviors better than anyone, it is important to include them as an integral part of the healthcare team when it comes to recognizing pain.² The change can be a loss of a normal behavior(s), development of a new behavior(s) for that individual cat, or a behavior problem.^{2,3,4,5,6} See Tables 1 and 2 for a list of changes in behavior and abnormal behaviors that can be a sign of pain.

Studies indicate that clients can often identify pain in their own pets more accurately than veterinarians can.^{3,4,5} Unfortunately, they often consider the changes to be associated with "old age" rather than pain or illness. To identify these changes, start by asking open-ended questions about changes in behavior or anything else since the last visit or the reason for the visit. A good example of the open-ended question is "What changes in behavior have you noticed?" Tables 1 and 2 indicate the most common behavior changes associated with pain.

The most common abnormal behaviors associated with pain are house soiling and aggression. Aggression may be human-directed or intercat aggression.

Veterinary teams and pain recognition and assessment

All team members should be educated to recognize pain for earliest assessment of discomfort during veterinary visits, or with surgical procedures or injuries. Client education for early pain recognition is critical as well. Although changes in behavior and behavior problems are common signs, they can also be associated with other conditions. Other parameters to recognize pain are body and facial posturing, mobility and other indicators.

Body or facial posturing

A painful cat may be tense on examination in an attempt to protect itself from pain. Often the cat will attempt to avoid lying on the painful area, and may crouch or be restless, moving from one position to another in an attempt to get comfortable.

The body may be hunched in pain with the back arched and the head lowered. A unique indicator of acute pain is half closed or squinted eyes.⁷

When assessing for pain following a procedure, compare posture and position in the cage post-procedure with what was noted prior to the procedure.

Changes in mobility may be the easiest signs to notice. However, most cat owners consider these to be normal aging changes instead of signs of degenerative joint disease. These include stiffness upon waking, legs that tremble or shake, being “down” in hocks or carpi, or a decrease in overall mobility. A common sign seen is the cat who wants to jump but hesitates, standing in position as if it is readying itself to jump but is thinking about whether it is worth the discomfort or effort.

Pain scoring should occur in all patients. There is now a validated acute pain scale for cats, and pictures and videos are available to go with that scale.⁷ A score that is more readily usable is also available, but has not been validated.⁸

Response to analgesia as a means of recognizing pain

It is important to note that fear or stress can also cause changes in behaviors, often making it difficult to differentiate between fear or pain, especially in the veterinary practice. For example, a cat that ‘freezes’ is signaling that it is fearful, anxious or uncomfortable. Physiologic signs such as increased respiration and heart rate, increased blood pressure, or dilated pupils may also be secondary to pain or stress or illness.

Even if cats do not express pain, it does not mean that they are not painful. *If there is a question regarding the presence of pain, administer an analgesic and assess the patient response. Response to therapy is an appropriate and important tool in pain assessment.*⁷

Preventing Pain

Handling feline patients to prevent pain

Because it is difficult to recognize pain, even before it is diagnosed it is important to handle each and every cat regardless of age as potentially painful. Since anxiety can exacerbate pain, allow the cat to hide in the bottom half of the carrier or a cat bed brought from home. Use gentle and respectful handling techniques. Non-skid surfaces prevent slipping. Allow the cat to be where it wants to be, and as comfortable as possible throughout the examination.

Start the examination from a distance to assess body posture, stance, and gait. If possible, entice the cat to walk but do not force it to do so. Usually the best way to assess gait is at the end of the appointment by placing the cat at the opposite end of the room from the carrier and watching the cat go to its carrier.

Examination should start with the least painful parts of the examination, and obtaining heart and respiratory rates as well as blood pressure prior to joint palpation improves accuracy of these results. If pain is noted at any time before or during the physical examination, stop and give analgesia, and examine the non-painful areas and collect lab samples prior to further assessing the painful areas. Transmucosal or intramuscular buprenorphine is an excellent analgesic in this situation.

There is potential for exacerbation of pain or further injury if cats with spinal pain, regardless of etiology, are held or picked up by the scruff, or other painful manipulations occur.

Many cats are uncomfortable due to DJD when legs are handled during examination or diagnostic testing. It is important that the cat be allowed to remain in positions it prefers, which are often more comfortable to them. Legs should not be stretched out tightly, but instead held in a comfortable position. Analgesia or anesthesia may be required prior to evaluation.

Weight optimization and prevention of dental disease

Preventive veterinary care can help prevent pain in the majority of our feline patients. Preventing dental disease, the most common condition seen in cats, prevents oral pain. Client education for home care and medical treatment to prevent dental disease is an excellent and cost-effective plan.

Obesity, the second most common condition in owned cats, exacerbate discomfort to joints. We know that weight optimization alone helps reduce pain in people and dogs with DJD,⁹ and it is likely that this is true in cats as well.

Preventing pain via peri-operative and “peri-procedure” analgesia

Systemic and local analgesics, including opioids, local and topical analgesics are part of analgesic protocols in feline surgical and dental patients. There are also many procedures that deserve analgesia prior to performing the procedure, such as anal gland expression, manual extraction of stool, ear cleaning, and radiographs. A complete list can be found in the 2007 AAHA-AAFP Pain Management Guidelines.

Home environment

Many cats have degenerative joint disease, and other cats may have difficulty getting to favored locations because of other medical problems. Providing ramps or steps to get to favored places, placing food, water, and litter in easily accessible places will allow cats to continue to perform their normal behaviors.

Preparing for the next visit

Hopefully medication prescribed for a chronic condition will keep the cat comfortable during future visits. Many patients with chronic pain have flare-ups of pain, and palpation can be uncomfortable. Burpenorphine sent home for transmucosal use and given approximately 30 minutes prior to the examination can be very helpful for patients that have had chronic pain demonstrated during veterinary visits.

Client education

Educate clients to watch for changes in behavior and to contact us if noted. Since cats that are painful often continue to eat and signs are subtle, there is another tip that can be eye-opening for clients. Ask them to put a picture of their cat on the refrigerator or elsewhere where they can see it frequently. Each year, put another picture up. When you see a difference, contact your veterinarian. Years go by and we don't notice the subtle changes – unless they hit us in the face. I learned this the hard way – after the fact with a beloved cat of my own.

Table 1. Changes in normal behaviors associated with pain

- Appetite
 - Decrease or increase
- Eliminations
 - Increase or decrease in volume
 - Changes in ability to get in and out of the box
 - Changes in how stool or urine is passed
- Grooming
 - Overgrooming in one or more areas
 - Not grooming +/- matting
- Sleep
 - Sleeping more
 - Sleeping less because cannot get comfortable
 - Restless
- Activity
 - Decrease or increase
- Vocalizing
 - Yowling during the night or at any time
 - Not meowing for treats or food as usual
 - Increase or decrease in purring
 - Purring can occur in cats trying to comfort themselves
- Play
 - Decreased
- Interactions with people or other pets
 - Intercat aggression
 - Human directed
 - Withdrawn or hiding
 - “Clingy”
 - More “cranky”

Table 2. Abnormal behaviors associated with pain

- House soiling
 - Urine and/or feces outside the litter box
 - May be over the litter box edge or in an area away from the box
- Irritable or cranky
- Aggression

- | |
|---|
| <ul style="list-style-type: none"> ○ Human directed ○ Directed toward another pet or pets |
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LITTER BOX BLUES

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Introduction

House soiling, which is often called ‘inappropriate elimination’ is the most common cause of surrender and euthanasia of pet cats.^{1,2,3} In fact, it is the most common cause of death in adult cats.⁴ The term ‘inappropriate elimination’ insinuates that the cat is doing something wrong, which is what most owners believe. House soiling is the preferred term because it means that the cat is urinating or defecating outside of the box without judgment of the behavior.

Although house soiling can also be frustrating for veterinarians, it helps to think about it as any medical case. In fact, an underlying medical condition is often the cause, either as the sole problem or in conjunction with another problem. Causes of house soiling can be divided into four major groups – medical problem(s), feline stress, marking, or litter box issues.

Diagnosis and treatment of house soiling keeps cats in homes, enhancing their welfare and the relationship with the human family. It also improves the owner’s relationship with the veterinary practice.

Setting the stage for the client

Many clients don’t see the need for diagnostics because their cat appears healthy. Instead, it is commonly thought that the cat is acting out of spite, with 65.8% of the cat owners that relinquished a cat thinking that it eliminated outside the litter box to spite them.¹ For example, the owner sees his or her cat urinate outside the box after the fiancé moves in and immediately assumes it is due to spite. It is often necessary to help owners understand that cats do not act out of spite but rather because the cat’s physical, social, or medical needs are not being met.⁵ In the case of the fiancé moving in, the cat may have had an underlying medical problem that was not noticed until now, or perhaps the cat experiences stress associated with the household change or the litter box not being cleaned as frequently – these are all potential causes but spite is not. A detailed history can help identify a cause, such as a change in litter, the litter boxes being moved to the basement, or the fiancé’s dog or cat also moving in.

History

Asking open-ended questions followed by more specific questions will yield a comprehensive history.⁶ Many owners do not seek veterinary care for house soiling, rather turning to the internet, a friend, or a pet store for advice. Increasing their awareness that we work with feline behavior concerns or refer the more difficult cases to a behaviorist supports owners and increases feline welfare. Asking about when the problem first started will often help owners remember a move, a vacation, construction, or another person or pet added to the household. The knowledge that something changed in the cat’s life, a species that likes a sense of control and predictability in their environment and is often fearful of change, are important clues.

Important questions are when the problem first started, how frequently it happens, and what is different now from when the problem first started. Often owners will note that the cat first

eliminated outside the box years ago, but it only happened once or twice. Often the cat has house-soiled in more than one home.

The problem may be related to another pet or person in the home, so it is important to ask about family members and all pets, as well as visitors. For example, in one case, the cat only urinated outside the box when the daughter came home from college with her 2 large dogs. Cats that are not bonded – recognized as never snuggling or grooming - are a common cause of stress and house soiling.

Ask about litter boxes, their size, shape, placement, and whether they are covered or uncovered. Many cat owners recognize that they need multiple litter boxes, but often put them in the same location, usually the basement. If a cat cannot reach the box, is another cat blocking the path or a dog is eating tasty treats from the litter box, or is the cat unable to make it downstairs anymore because of degenerative joint disease or another medical problem?

Asking owners to provide a simplified floor plan with location of litter boxes as well as other resources (e.g., resting areas, food, and water) can help identify problems that might not be recognized otherwise. This can occur at the first appointment but often is done as a component of a behavior consultation once medical problems have been ruled out. Pictures of litter boxes and videos of the cat using the box or an alternate location are also helpful.

People often are attracted to litters that will mask the scent of urine and feces, but the scent may be offensive to the cat. The owner may prefer crystal or pine or another substrate, but what is important is what substrate the cat prefers.

Cats are fastidious animals, but many owners don't scoop litter boxes daily, and boxes may not be cleaned completely for many months or longer.

Other history is also important, including the cat's ability to jump and climb, vomiting, appetite, and interactions with others in the household.

As with any medical problem, history is an important piece of the puzzle. However, even if household stress is present, there may also be a concurrent medical problem leading to the house soiling.

Physical examination, differential diagnosis, and diagnostics

A comprehensive examination will identify changes in weight, assess body and muscle condition scores, and include an orthopedic evaluation. With an increase in older cats due to advances in medical and home care, many of our patients have degenerative joint disease or other causes of difficult mobility (e.g., diabetic neuropathy, hypokalemia or other causes of weakness or pain). Hyperthyroidism is a common cause of fecal soiling, but may lead to urine soiling as well. Any urinary tract or gastrointestinal disease may lead to house soiling.

Diagnostic testing should always include a urinalysis, complete blood count, and chemistry profile for urine soiling. In addition to these tests, fecal tests should be done for cats that are

house soiling. Thyroid testing should be performed in cats 7 years and older, or if they have other signs consistent with thyroid disease.

Radiographs and abdominal ultrasound are also needed in many cases if obvious answers are not found with baseline testing.

Medical etiology

Treatment of specific medical conditions should occur in conjunction with making litter boxes easily accessible and more appealing to cats, in addition to providing them space where they don't need to interact with other animals to reach the boxes. Even if the medical problem is the primary cause, the cat may have found a preferable area to eliminate while ill, and this must be addressed as well.

Feline idiopathic cystitis

There is a strong link between feline stress and the chronic pain syndrome, feline idiopathic cystitis (FIC).^{7,8,9} Also called feline interstitial cystitis, it is the most common cause of feline lower urinary tract disease, with 54-64% of cats presenting with lower urinary tract signs having idiopathic disease.¹⁰ FIC was initially considered a disease of the bladder alone, but it is now recognized that the response is activated in the brain by the hypothalamic stress response system.⁸

Stressors include unfamiliar environments and individuals, and a lack of predictability and sense of control, either in the home or the veterinary practice. For example, a hospitalized cat may have a perception of poor predictability and a lack of sense of control if there are inconsistencies in caretakers, feeding and cleaning routines or periods of light and dark.⁸

A significant decrease in the frequency of FIC signs has been seen with environmental enrichment, familiarity, and a sense of control.^{7,10} Based on this information, veterinarians can help cat owners recognize environmental stressors and how to improve the environment and predictability (a means to providing a sense of control) for the cat.

Marking behavior

Marking behavior is common in unneutered cats, but may also occur in neutered cats. Marking is a means of feline communication, and includes urine marking (spraying), fecal marking (middening), rub or cheek marking, and scratch marking. Cats communicate through scent marking and body posturing to avoid conflict and protect self. Marking in neutered cats usually indicates a stressful environment.¹¹ Providing a safe environment with easy access to litter boxes reduces stress. Synthetic feline pheromone analogs also increase security in the environment. Anxiolytic medications may be needed temporarily or in some cases for extended periods depending on the household situation.

Environmental problems

Litter boxes

Most commercial litter boxes are too small for cats, with a good length being 1.5x the length of the cat from the tip of nose to the base of the tail. This allows the cat sufficient space to step into the

box, turn around to dig a hole, eliminate, and then cover (not all cats cover). Preferable are large storage containers and dog litter boxes. An opening can be cut out of the front of a high-sided plastic container to allow easier access for cats having difficulty jumping over the edge. High sides are needed for cats that are “high risers” or spray in the litter box.

Location, location, location

What may be more important than the number of litter boxes is the location of the boxes. Most people keep litter boxes in the basement and all right next to each other. They think they have 3 boxes, but from the cat’s perspective there is only one box if they are all in the same location. Instead, boxes should be separated from each other, and with a visual barrier to allow privacy and safety for a timid cat.

If a cat is fearful of something blocking its pathway –such as another cat it doesn’t like staring at him or her from the staircase or hallway to the litter boxes - the cat is likely to find a safer place to eliminate. Having at least one litter box on each floor is ideal, especially for cats that have more difficulty going up and down the stairs.

Cats are not the only ones who may block access to a box. If there is also a dog in the home, try to place litter boxes in places that are still easy access for the cat but difficult for the dog to get to. People may also inadvertently frighten a cat from using a box, such as an active or loud child, an adult trying to medicate the cat while in the box. Fortunately, with client education and boxes placed in different locations in the home, the cat can choose which path to take and remain safe.

The fastidious feline

Cats are extremely clean animals, and they do not want to eliminate in dirty boxes. Litter boxes should be scooped at least once daily. They also need to be cleaned out at least once weekly with non-scoopable litter and every 2-4 weeks with scoopable litter. Before replacing litter, use mild detergent or just hot water to clean the box and dry thoroughly. Having additional boxes allows one to rotate in a clean box while another is being cleaned and dried.

For cats with polyuria or on subcutaneous fluids, more frequent scooping and cleaning is necessary. This is also indicated for cats with diarrhea.

Litter types

The pet stores have numerous types of litters marketed for humans. Many contain deodorizers, and may be made of pebbles, crystals, pine, corn, or paper. In the wild, cats use sand or dirt, and most cats prefer unscented sand litter.

Cleaning areas of house soiling

Many enzyme breakdown products are available on the market, but many are not effective. Anti-Icky Poo and Urine Off are excellent products to eliminate the smell of urine outside the box so that cats are not attracted back to that area.

Preventing house soiling

It is always easier to prevent house soiling than to treat it. Unfortunately, assumptions are often made that owners know how to purchase litter boxes and litter, and where to put the boxes. Nothing can be further from the truth. Providing cat owners with information on litter box size,

types of litter, frequency of box cleaning and how to clean the box will help prevent problems. Educating owners to contact the veterinarian even if the cat misses the box just once will help them recognize that we know how to deal with behavior as well as medical problems.

Conclusion

Behavior problems, and especially house soiling, are a major concern to owners. Working with owners to identify the cause and resolve the problem enhances the relationship that clients have with us and their feline family members.

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ENVIRONMENTAL NEEDS OF INDOOR AND OUTDOOR CATS

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Introduction

Advances in feline medicine have increased the pet cat's physical health and longevity¹, but emotional and environmental health needs often go unrecognized. As the indoor cat population has grown, this species has suffered from boredom, stress associated with inadequate environments, obesity, and diabetes mellitus.^{2,3}

Behavior problems and normal feline behaviors that people consider undesirable can also occur when cats' needs are not met. Environmental stressors can even lead to physical health problems, such as feline idiopathic cystitis. These problems occur due to the disparity between who cats really are and the impression that many owners have, which is that they are low maintenance and easy to care for pets.

The cat is a paradox – although fairly adaptable and social animals under the right conditions, cats have retained many of the behaviors of their wild ancestor, *Felis lybica*.^{4,5} In fact, pet cats are still more similar to their wild ancestors than to other species and require an environment that provides for their needs. Understanding the cat, its normal behaviors, and its needs can often prevent or resolve stress, boredom, and behavior problems. Regardless of the age and physical health of the cat, and regardless of whether the cat is indoors only, indoor/outdoor, at home or at the veterinary practice, providing for the cat's environmental needs is not optional but rather essential for its welfare.³

Feline Welfare and the Veterinary Responsibility

Within the past decade, a large number of worldwide veterinary organizations have rewritten their veterinary oaths to emphasize welfare. Animal welfare is defined by the AVMA as: "...how an animal is coping with the conditions in which it lives. An animal is in a good state of welfare if (as indicated by scientific evidence) it is healthy, comfortable, well nourished, safe, able to express innate behavior, and if it is not suffering from unpleasant states such as pain, fear, and distress. Protecting an animal's welfare means providing for its physical and mental needs." Many feline patients are not allowed to express innate behaviors, often leading to fear and stress - to poor welfare.

Understanding the cat and its needs

Cats as solitary hunters

Because cats are solitary hunters of several small prey per day, they must maintain their physical health and avoid danger. They do so through two major protective mechanisms – territoriality to maintain safe space and having a heightened fear response. Familiar territory provides cats with a sense of control over their physical and social environment.⁶ Having a sense of control - even if it is not exerted - makes the cat more comfortable and reduces stress.⁷

A primary goal of communication between cats is to protect territory and avoid physical altercations.⁸ Cats communicate through body and facial posturing, as well as via their senses. Scent marking is most important for cats, with scent and pheromonal signals used as distance communication to keep other cats away without the need for physical contact. Scent marking occurs via facial and body rub marking, scratching, urine spraying, or middening (fecal marking). Spraying in neutered cats is usually secondary to stress in the environment.

Cats possess a heightened fear response as a protective response to fear.⁹ If cats are forced to leave their familiar territory or a threat enters their territory, they respond to this confrontation by avoidance or hiding, with fighting only occurring if there are not other options to protect self. Fear commonly occurs when a cat is taken outside its environment and brought to the veterinary hospital. Providing a place to hide for both inpatients and out-patients can prevent fear-associated aggression.

Providing choice in the environment through multiple resources - hiding, perching, feeding, water, and toileting areas - in multiple locations in a multi-cat household will reduce fear and provide cats with a sense of control and more secure environment.³ This is important regardless of whether it is the home environment, veterinary practice, cattery, or shelter.

Feline environmental needs

Safe space

Hiding is a coping behavior that cats often display in response to changes in their environment.¹⁰ In the home, this could be an unfamiliar person or pet. Problems often occur with a newly adopted cat being introduced to already existing household cats without gradual introduction. Even if it is not a newly introduced cat, it is not unusual for cats that live in the same household not to like each other and choose to rest in a safe place away from others. Even affiliate cats – cats that like each other – prefer to sleep alone and out of sight of others approximately half the time.⁶ Appropriate sleeping areas are also good hiding places, such as a box, a cat bed with high sides, or a carrier with soft bedding such as fleece.

In the veterinary hospital, a safe place is necessary for both in-patients and out-patients. The carrier – especially if the cat has positive experiences and familiarity with it in the home environment – is an excellent safe place. Allowing the cat to rest in the bottom half of the carrier during examinations and providing either the carrier or another hiding place during hospitalization or boarding will increase feline safety and security, and decrease fear-associated aggression.

Elevated resting areas

Increasing overall space by providing cat trees, perches, shelves, or other vertical space helps prevent conflict between cats.³ Cats can also monitor or oversee the environment from a vertical space.

Scratching

Scratching is a normal feline behavior that marks territory with both scent and visible markings. It also is done to sharpen claws, remove old sheaths, and to stretch muscles. Providing scratching posts with preferred texture, such as sisal rope or natural wood, as well as in multiple locations helps prevent furniture destruction. Posts should be placed in locations where cats prefer to scratch – usually next to a most prominent piece of furniture, but sometimes also where new scents occur (e.g., the front or back door).

Normal feeding behavior of the solitary hunter

Cats are not pack hunters, but rather solitary hunters, eating 10-20 small meals per day, with repeated cycles of hunting to catch their small prey. Not all attempts to catch prey are successful (some suggest that up to 50% of the hunt cycles are not successful).¹¹ Think about how much time and energy the cat

utilizes just to survive!

Compare that to what happens with many owned cats. People usually control the feedings, often providing 1-2 meals daily of highly palatable food. The inability to control access to food is associated with feline stress.¹² The sedentary house cat expends very little energy and time hunting, and more time eating. In some countries, including the US, many cats are kept indoors. Whether to protect the cat itself or wildlife, failure to provide opportunities for predatory behavior may deprive cats of mental and physical activity, and may contribute to development of obesity and other health problems.^{11,13}

Because people are social eaters usually enjoying meals together, they often provide multiple cats with food either in one bowl or in bowls placed side-by-side, not recognizing that this causes competition for food resources and stress for the cat. One can understand why some cats may eat large volumes very rapidly, often overeating, and perhaps regurgitating. Stress is usually the short-term result, and obesity and obesity-associated diseases are more long-term outcomes.

Regardless of how much cats are fed, the hunting instinct still exists; cats often bring in these unwanted “presents” to their people. Cats are also crepuscular animals, hunting primarily at dawn and dusk, when their prey is usually present. This sometimes leads to waking owners during the wee hours of the morning, which can be quite annoying for humans. Often owners inadvertently reinforce this behavior in their attempt to quiet the cat so that they can go back to sleep, leading to a long-term and frustrating problem for owners. Client education can prevent this problem as long as we welcome clients to discuss their frustrations or concerns about their cats with us.

As veterinarians, we have the opportunity and responsibility to educate clients about normal feeding behavior of the cat as part of the nutritional advice we provide. This will help prevent both medical and behavioral problems, obesity, and stress in the home environment. This can be done by simulating “hunting” through the use of food toys or puzzles, tossing kibbles, or hiding them around the house. This more normal feeding behavior will increase exercise, reduce boredom, and help prevent obesity.¹⁵ Providing feeding areas in multiple locations which are out of sight of each other will prevent competition for food resources.

Drinking behavior:

Cats in the wild drink water in locations separate from food. Some cats prefer running water, and some still water. Provide water dishes in multiple locations and away from food.

Play behavior

Queens teach kittens to hunt through play behavior. The rough tussle and tumble of kittens help them hone their hunting skills. Kittens and even adult cats, especially if housed singly, may want to play with their owner’s hands and feet in the same way. When young, people often think this is cute, and unknowingly reward the behavior. Play aggression can lead to human injury and zoonotic disease.

Playing with an interactive toy minimally once to twice daily can prevent this problem. Cats learn to anticipate and prefer the routine of playing daily at a certain time, even if toys are rotated. Cats playing together also provides an outlet for this behavior. Self play is also important, and can be provided through puzzle feeder toys or other favored toys (e.g., hair scrunchy, foil or paper ball).

Toileting areas

Litter boxes should be placed in multiple locations around the home, but away from food, water, and

sleeping areas. It is not uncommon for cat owners to prefer to place 2 or more litter boxes in the basement next to each other. This poses multiple problems – usually there are noisy appliances and equipment in the basement, the boxes next to each other don't provide easy access to a box if a more confident cat is blocking a timid cat, and a cat with degenerative joint disease or another condition making it difficult to get downstairs – that can lead to a cat soiling outside the litter box.

Many litter boxes are also too small for cats. Cats prefer larger boxes so that they can turn around, dig, and eliminate. Boxes should be 1.5 times the size of the cat from the tip of the nose to the base of the tail. Dog litter boxes and plastic storage containers with an opening make excellent cat boxes.

Some cats will eliminate in any type of litter. Others prefer a soft consistency, such as sand over pebbles. Deodorizers or scented litter since as pine are developed to attract consumers and not the cat. Some cats find them offensive. Most cats do well with unscented clumping sand litter.

Scooping boxes a minimum of once daily and changing boxes completely when needed (weekly for clay or non-clumping litter and once every 2 or more weeks for clumping litter) will also help to prevent house soiling problems.

Single vs. Multiple Cat Households

Cats are social animals, but their social system is flexible, meaning that cats can live alone or in groups called colonies if there are sufficient resources.^{5,11} A single cat must have its environmental needs, including play, met through both self and interactive play with a family member they are comfortable with. For some cats, that is a dog or other pet, or any person in the family. For those that have not been well socialized, it may only be in a quiet place with the one person they like.

In a multiple cat household, each resource must occur in multiples, with easy access and out of view of other resources. This includes hiding places and use of vertical space to allow cats to be apart if they so choose. Vertical space increases overall space and provides for the cat to oversee the environment. Litter boxes, food, and water stations that are placed in different locations so that individual cats don't need to see each other reduces competition for resources, bullying, and stress.¹² Even cats that demonstrate affiliative behavior, such as rubbing against or grooming each other, or sleeping in close physical contact prefer to rest by themselves.

Cats usually do not readily welcome unfamiliar cats. In a study of 1,286 relinquished cats, relinquishment was associated with the number of pets in the household, as well as new cats being added into the home environment.¹⁴ Introductions must be gradual, and with each of the resources available in multiple locations to prevent the need to pass an unfamiliar cat.

Just because cats come together for feeding or to sleep on the same bed, it doesn't mean that they like each other or that stress isn't occurring in the feline household; in many households, cats come together because the primary resources are placed in one location. Since cats are more likely to rest or sleep alone, multiple comfortable resting areas should be provided. Inter-cat conflict and behavior problems often occur because household cats don't have multiple resources in multiple places, and therefore cannot avoid the other cat(s).

The Outdoor Cat

In some countries, veterinary behaviorists consider it inhumane for cats to be indoors only. In the

United States, many veterinarians recommend an indoor only environment. Watching videos of the active cat outdoors or even the cat that sits and watches nature certainly demonstrates the enrichment difference between many indoor and outdoor cats. However, these environments are also fraught with challenges. The territorial cat wants the safety of its environment that may be challenged by other outdoor pet cats or feral cats. There are the potential dangers of viruses, vehicles, and toxins, in addition to wounds from cat fights.

One may consider controlled outdoor exposure as the best alternative, either walking the cat on leash or having the cat stay in an outdoor enclosure. With cat enclosures, it is still essential to meet the cats' environmental needs, with perches and resting areas provided.

Meeting the environmental needs in a caged environment

Feline environmental needs must also be met when housing cats in a veterinary practice or shelter regardless of the length of stay.¹⁵ These include a resting or hiding place, perch, litter box, food, water, and play. Cats that like attention should also receive human attention. Both the size and complexity of the cage are important to meeting the needs of the caged cat.¹⁵ Cages can be enlarged by adding vertical space or combining 2 smaller cages. Cages should be warm, non-slippery, and without the cat seeing its reflection or other cages. Tall cat beds, a cardboard box, or the cat's carrier make comfortable hiding places.

The old rules of cleaning the cage completely once or twice daily are no longer the best way. Cats mark their territory with facial pheromones, which makes them more comfortable in the environment, and it is important to avoid cleaning these marked areas until the cat goes home or to a home. It is best for the cat to remain in the cage for the duration unless it is soiled to the point that it cannot be cleaned without moving the cat to another cage. The preferred method is to spot clean the cage if it is not soiled.^{16,17} Spot clean or clean around the cage with minimal disruption of the patient. Do not spray areas clean, but wipe them. Avoid wiping areas that are not soiled. Additionally, the towels or blankets in the cage should not be changed unless soiled, because doing so takes away the familiar scents and introduces unfamiliar scents. The same goes for litter boxes: Try to keep the same litter box with the cat during the cat's stay, scooping two or more times daily, but not washing out unless necessary.

Conclusion

When we understand cats and meet their environmental needs, regardless of at home, indoor or outdoor, in single or multiple cat living situations, we enrich their lives and increase their welfare. Providing the environmental needs in hospitalized, boarding, or shelter cats is also essential and prevents fear and fear-associated aggression.

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CANINE AGGRESSION: RISK ASSESSMENT, PROGNOSIS, AND SAFETY

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Before implementing a treatment program for canine aggression, a behavioural consultation is required to determine the diagnosis, prognosis, and what must be implemented to safely manage the problem. Prevention of further repetition is essential to insure safety as well as to prevent further aggravation of the problem which is further conditioned each time the dog is exposed to the stimulus with an unpleasant outcome, and each time the pet is negatively reinforced by removal of the stimulus. The different presentations of aggression, including stranger directed, owner directed, aggression to strangers, owners, unfamiliar dogs, family dogs and pain induced, which may have different underlying mechanisms.^{1,2} In a recent study of veterinary behaviour cases, owner directed aggression was the most frequent complaint (39%) and aggression to unfamiliar people (22%) with 1.4 diagnoses per pet.²

Prognosis

Prognosis is about both safety and the potential for improvement. The ESVCE position statement on risk management (esvce.org) describes the following steps; identify risk factors, determining who might be harmed and how, discuss precautions for each risk, record and implement, and update and review

The initial focus must be on safe management and prevention of further aggression. It is essential to insure that owners have realistic goals as to what can be achieved and how this can be accomplished. Rehoming or euthanasia may be necessary if owners are unwilling or unable to implement safety strategies or accept the limitations of what might be achieved.

Family and environmental factors including presence of children, mentally or physically challenged, understanding and commitment and the limitations of the household are critical factors in assessing risk and in determining prognosis. Unpredictability, dogs that bite in response to benign stimuli, dogs greater than 18 kg and dogs aggressive to family members are risk factors for rehoming or euthanasia.⁶ Source of dogs, age of acquisition, age of onset, breed, early environment, and medical health all influence the development of aggression and whether it can be effectively managed and treated. Unpleasant experiences by the owner (emotional state, actions) or the stimulus (threat, aggression, fear) will condition further fear. Successful removal of the stimulus with aggression or removing the pet when aggressive (although necessary for safety) negatively reinforces the behaviour.

Predictability: The most critical issue is to identify each stimulus (trigger) and situation in which aggression may arise to implement safe preventive strategies. Unpredictability is a risk factor for euthanasia.³

The bite: Dogs that threaten before biting, inhibit their bite or try to avoid, have a better prognosis provided the owner can recognise dog signalling and identify each situation in which the dog might bite. Dogs that bite intensely in response to benign stimuli are at greater risk.³ Bite scales may help to evaluate severity. <http://avsabonline.org/blog/view/ladder-of-aggression>

Learning: Aggression that is longstanding may be more resistant to change, in part because of repeated conditioning and learning. When aggression results in successful removal of the stimulus, the behaviour is negatively reinforced. In addition removal of the pet from the situation during the aggressive display, (although necessary) negatively reinforces the behaviour. Unpleasant experiences condition further fear including a) the owner's emotional response (fear, anxiety), b) actions (confrontation, punishment) or aggression, and c) fear or threats (real or perceived) from the stimulus.

Environment: Does the environment provide practical options for preventing access to aggression evoking stimuli and making graduated improvements?

Family limitations: When children are at risk the prognosis is more guarded since they are more vulnerable; more unpredictable, and less able to recognise and react to dog signalling. If there is a history of child aggression in the home, keeping the dog may not be advisable. However, 66% of dogs had never previously bitten a child and 19% never bitten an adult. Familiar children are most frequently bitten during resource guarding or engaging in petting or hugging especially when they initiate approach. Unfamiliar children are most often bitten on or in the vicinity of the property whether or not they interact.⁴ In addition, the prognosis / risk is greater when there are people in the home who are unable to interact appropriately and recognize signalling due to mental or physical disabilities.

Dog / signalment / breed: Dogs aggressive to family members are at greater risk for euthanasia if greater than 18 kg.³ Male dogs appear to be over-represented.² Age of onset may also be a risk factor. Food guarding, fear and anxiety in dogs 2 to 4 months of age is predictive of adult fear and aggression.^{5,6} Studies have demonstrated breed differences; Golden Retrievers were at lower risk for aggression, guarding breeds at a greater risk for stranger related aggression, and some breeds showing greater propensity for owner directed aggression.⁷ In the English Cocker and English Springer Spaniel, aggression with impulsivity was associated with altered serotonin or its metabolites indicating a likely genetic propensity.^{8,9} There may also be a link between coat colour and aggression in blonde cocker spaniels.⁸ Since breeds have been selected for a variety of functions associated with aggression including guarding, herding, and hunting, breed differences should not be surprising. However, it is not the breed but rather the individuals within a breed that display aggression consistent with breed function.

Background / Source: Dogs obtained from pet stores, puppies raised in non-domestic environments, and a lack of urban experience from 3 to 6 months of age increases risk of aggression.¹⁰ Studies have also demonstrated that breeding for conformation may be associated with greater social fear and greater aggression to family members than working bred dogs.^{8,9}

Medical and behavioural pathology: Animals with chronic or recurrent illness may be difficult to manage. A history of a pruritic skin disorder or steroid use has been associated with aggression.⁵ In another study, medical conditions were suspected in 50% of dogs with aggression to children.⁴ Aggression that is behaviourally pathological may have a guarded prognosis, pending response to medication.

Compliance: The family's expectations, commitment, and ability to comply are a major factor in whether the dog can be safely kept in the home. Some families are unwilling or unable to

manage the risk, while others have unrealistic goals for a quick fix or greater improvement than is practical.

Safety and Management strategies

Strategies for effective and safe management include preventing access to the stimulus, identifying and avoiding triggers, understanding canine communication, and physical management devices.

1. Stimulus avoidance: Each stimulus and situation that might incite aggression must be identified to implement strategies to prevent, avoid, move the dog far enough away or redirect the dog into desirable outcomes. Each recurrence is not only a safety issue but also a learning experience that will further reinforce or aggravate the problem. In addition, identifying every stimulus and how it can be effectively muted or minimized and graded for exposure is essential for behaviour modification (desensitization and counterconditioning).
 - i) Physical separation will insure that the pet cannot see, hear, or access the stimulus (person, animal) and the stimulus cannot access the pet.
 - ii) Dogs that are aggressive with visitors to the home should be confined when visitors arrive. This could be to a separate room, a crate or pen, in the yard, or with a tie down but ideally should be the dog's safe haven. "A safe haven is an area where the animal is in control and which has become a conditioned place of safety outside of times when there are significant stressors. Consequently, when the animal is faced with potential stressors it can retreat to this place and feel relatively safe."¹¹ Alternately the dog might be kept leashed under control of an adult family member at sufficient distance (sub-threshold) from the stranger. If the triggers are specific individuals (e.g. children), times, or places, then avoidance strategies would primarily be necessary to these stimuli.
 - iii) When dogs are aggressive on walks, stimuli can be avoided by limiting walks to places and at times where stimuli can be avoided or by keeping the dog on property with alternative forms of enrichment (yard play, food manipulation toys, nose work). Alternately it might be possible to maintain sufficient distance from the stimuli, or to move far enough away for the dog to settle, should signs begin to emerge.
 - iv) Any type of approach, handling, or contact that might incite fear, anxiety or aggression should be avoided. Dogs that become aggressive when resting or in possession of food should be confined at these times (safe haven). Dogs that are protective of other resources (e.g. toys, garbage, stolen items) should be housed away from these resources or muzzled at any time they cannot be effectively supervised (with leash control if necessary). Offering a highly valued resource might be sufficiently enticing for the pet to give up a resource or leave a resting area.
 - v) For any interaction or procedure that cannot be avoided, the dog should be managed with leash and head halter, muzzle or under sedation.
2. Owner responses: Owners must cease all actions and interactions that might incite fear or aggression. Confrontation, corrective, and punitive techniques are counterproductive and will increase fear and uncertainty. Punishment may also suppress the threats that precede aggression or might cause a passive dog to become offensive. In addition when people are fearful or angry, the dog's anxiety is likely to be heightened.

3. Reading body language (See resources below): Owners should have a sound understanding of canine communication and signalling to be able to recognise facial, body and tail positions of fear, anxiety or conflict as it first begins to arise.
4. Behaviour management products: A leash and head halter that controls the muzzle can be used to refocus the dog's attention or reorient the dog away from stimuli that might incite aggression, prompt desirable outcomes (e.g. sit, back up, walk away) and close the mouth in an emergency situation. Alternately a leash and front control harness can provide added control to calmly and effectively move the dog away from potential problems. Visual and auditory stimuli might be muted using eye or ear covers, music, or white noise. A basket muzzle provides safety and a means of calmly managing the situation. When using any of these products care should be taken to condition a positive association before using for training and exposure. A Treat and Train provides an additional option for immediately rewarding and shaping desirable outcomes at a location such as on a bed or mat.
5. Drug therapy and natural supplements might be indicated for reducing fear, anxiety, impulsivity or reactivity and control underlying behavioural pathology.

Foundation exercises: Training should focus on teaching those behaviours that will be required to manage and improve problems, beginning in environments where successful outcomes can be achieved, safety can be insured and fear evoking stimuli avoided. Highest value rewards should be used to train and shape progressively more calm / settled responses. Indoor training should include sit/watch, down/settle, go to a mat or crate, and perhaps give/drop, leave it and come (depending on the problem). For problems on walks, training should include sit / watch, loose leash walks, and turning to walk away or backing up. These cues might then be used for successful and positive control for desensitization and counterconditioning and response substitution in the presence of the stimuli (See notes on treatment of fear aggression).

Body language and safety

Dr. Sophia Yin: drsophiayin.com, drsophiayin.com/resources/video_full/dog-bite-prevention-psa-why-dogs-bite-and-what-to-avoid

Learn to speak dogs and teach your kids: doggonessafe.com

Modern Dog Magazine: <http://www.moderndogmagazine.com/articles/how-read-your-dogs-body-language/415>

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Zoom Room Guide to Body Language: [youtube.com/watch?v=00_9JPtXHI](https://www.youtube.com/watch?v=00_9JPtXHI)

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CANINE FEAR AGGRESSION TREATMENT – MEETING PEOPLE AND DOGS

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Aggression directed toward unfamiliar pets or people is predominantly a problem of fear and/or anxiety. In addition, when aggression is limited to, or is more intense on the home territory, there may be a territorial component. A correlation has been found between aggression to unfamiliar people and dogs that were raised in non-domestic environments (e.g. kennel, garage, barn), and a lack of experience with urban environments between 3 and 6 months; however, no association was found between environment, urban experience and aggression to other dogs.¹ Aggression may be due to medical or behavioural health issues such as impulsivity with alterations in serotonin in English Cocker and Springer Spaniels.^{2,3} In addition dogs on a leash are reported to be twice as likely to threaten and bite.⁴ This may be due to an inability to escape or control access to stimuli, restricted opportunity for normal communication, owner influence, and previous experience. Treatment requires safe and effective management of the problem with preventive measures; training to achieve focused and calm behaviours in the absence of any stimuli; teaching cues that communicate to the pet to focus and relax; management products; drugs or natural products where indicated and graduated exposure training (desensitization and counterconditioning and response substitution).

Diagnosis

Underlying medical problems that might cause or contribute to the signs must first be ruled out or (e.g. neurologic disease, endocrine disorders, metabolic disease, pain). The diagnosis, prognosis (risk assessment) and treatment plan, will then be determined from the history, evaluation of the pet and viewing any movie clips the client can provide.

Prognosis

Prognosis is about both safety and the potential for improvement. (See esvce.org for position statement on risk management). In one study of dogs that were aggressive toward unfamiliar dogs, 76% could be around other dogs on leash outdoors after treatment.⁵

The initial focus must be on safe management and prevention of further aggression. It is essential to insure that owners have realistic goals as to what can be achieved and how this can be accomplished. Rehoming or euthanasia may be necessary if owners are unwilling or unable to implement safety strategies or accept the limitations of what might be achieved.

Safety and Management strategies

Strategies for effective and safe management include preventing access to the stimulus, identifying and avoiding triggers, understanding canine communication, and physical management devices.

a) Stimulus avoidance: Each stimulus and situation that might incite aggression must be identified so that it can be prevented or avoided. For dogs with aggression outdoors / on walks, stimulus avoidance can be achieved by avoiding dog parks or specific locations, avoiding walks, walking the dog when and where stimuli can be avoided or insuring that sufficient distance from stimuli can be insured. Physical products including leash and head halter, leash and front control harness or with muzzle attached can help to insure safety. When dogs are aggressive to visitors coming to the home, the dog can be confined to or tethered in a location where it can be safely

and comfortably housed, or kept on leash under the control of one of the owners. If specific stimuli, times, or locations can be identified (children, visitors, food bowl, dog park) only those might need to be avoided.

b) Owner responses: Owners must be aware of how their actions and interactions influence behaviour. Confrontation, corrective and punitive techniques, or attempts to control through dominance are counterproductive and may increase fear and aggression.^{7,8} In addition when owners are fearful anxious, the dog's anxiety is likely to be heightened.

c) Products, drugs and supplements might include i) a safe haven / secure confinement, ii) leash and head halter or leash and body control harness (not prong, choke, shock), iii) basket muzzle, iv) products that mute visual and auditory stimuli such as eye or ear covers, cage covers, music or white noise and v) drug therapy and supplements

Reading body language

Owners must know when and where aggression might arise, and have a clear understanding of canine communication to be able to recognise facial, body and tail positions of fear, anxiety or conflict as it first begins to arise including tense, raise hackles, ears flattened, licking lips, yawning, and panting. This is essential for safety and prevention, to effectively address pet welfare, and to understand the threshold / limits for safe and effective exposure training (response substitution, desensitization and counterconditioning). See resources below.

Structure and consistency

All interactions should be structured and predictable. Before giving anything of value the dog should be consistently taught to sit calmly (say please by sitting, structured interaction training, learn to earn, nothing in life is free, no free lunch). The dog should be taught to sit or lie down calmly before getting anything of value, most specifically when seeking attention or affection but also before play (throwing a toy for fetch, nose work), putting on and taking off leash, going out the door for walks, meeting and greeting and giving food or treats. Gradually shape calmer and more focused behaviours. Consistent and predictable interactions remove anxiety, uncertainty and arousal, by using rewards solely as a reinforcement for desirable behaviour, and giving the dog control of its resources by sitting calmly. See handouts and videos at drsophiayin.com

Foundation Training

a) During the preventive program, the owner should teach the foundation behaviours needed for achieving the desired outcomes during future exposure. Behavioural guidance should focus only understanding and learning how to implement positive methods of behaviour modification with resource material and the support of a force free trainer. The guiding principle should be "reward what you want and ignore or prevent what you don't want." Confrontation, corrective, and punitive techniques or approaches that are intended to control through dominance are counterproductive and might further compound fear and aggression.^{7,8} Products that might be utilized to help insure safety (e.g. muzzle) and better achieve training goals (e.g. head halter) should be gradually and positively introduced in advance of the exposure training. In addition, any drugs or natural products to control underlying pathology and reduce anxiety and reactivity should be administered to achieve optimal therapeutic effect in advance of exposure training.

b) For foundation training, the dog will first need to be taught the behavioural responses that will be used to calm the dog in the presence of unfamiliar dogs and people including sit/watch, loose leash walk, backing up, turning away, and going to a mat or bed on cue (safe haven).

Come or leave it, down/settle, and drop/give might also be appropriate. Target training, clicker training, and the use of a leash and head halter or leash and body control harness can be useful management aids. Highest value rewards and clicker training should be used to shape gradually longer and more relaxed responses (body postures, breathing).

c) Training should begin in situations and locations where the pet is calm and focused, using valued rewards to achieve desirable outcomes before gradually progressing to other environments with increasing distractions. The owner can then focus on training the dog to calm in the presence of familiar people or dogs as it begins to become aroused or excited. The use of a leash and head halter can help to provide physical control and safety for maintaining focus or turning the pet away when potentially problematic situations arise.

Exposure Training

1. Once foundation exercises are reliably trained, drugs and/or natural products have reached efficacy and the situations in which problems might arise have been successfully managed, the owners might proceed to controlled exposure training. A gradient of stimulus intensity, reward gradient, desensitization and counterconditioning, and response substitution should be reviewed. Most owners will benefit from the oversight and guidance of a reward based trainer or behavioural technician for recognizing canine communication signalling, identifying the behaviour threshold and implementing the exposure program.

2. Stimulus exposure: The goal is to get calm and positive outcomes during stimulus exposure while working at or below the threshold at which the dog exhibits anxiety. Training should begin at or below the threshold at which the dog exhibits anxiety. Exposure should be set up (set up to succeed) by controlling all parameters (stimulus, dog, environment) and associating favoured reward with each exposure to the stimulus. Desensitization and counterconditioning is achieved by repeatedly exposing the dog to stimuli at level below the threshold and pairing with high valued rewards to make a positive association. Calming the dog with commands, “attitude” and the aid of a device for additional control if appropriate, is response substitution.

3. Stimulus gradient: Controlling the stimulus and introducing it in a graduated manner can be a challenge to implement. Stimuli might be visual, auditory, olfactory, or tactile. Stimuli can be controlled in intensity by a) distance, b) location c) stimulus characteristics (e.g. uniform, height, age, sex, breed, etc.) d) activity (e.g. motion, volume) and e) by exposing to individual components of the fear evoking situation one at a time. Video or TV images might also be a starting point.

Outdoors the stimulus could approach slowly while maintaining an acceptable distance or walk past slowly and calmly parallel to the dog. Indoors the dog can be brought out from confinement to maintain a distance where it can be successfully calmed and rewarded (response substitution) or high value rewards paired with each exposure (countercondition).

With further exposure the stimulus can move closer, increase movement, or volume intensity or activity. Throughout the training owners must be observant of any signs of fear or anxiety to be able to stop the exposure and reduce the intensity of the stimulus (or remove the pet) to end on a calm, positive outcome.

Drugs and Supplements

See notes on drug therapy for aggression in dogs.

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Resources

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2. Web resources: www.drSophiaYin.com, www.clickertraining.com
3. Canine communication and body language:
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<http://veterinarynews.dvm360.com/do-dogs-appease-each-other-or-us-veterinary-research-focuses-watching-and-labeling-canine-interactions>
 - Learn to speak dog and teach your kids: doggonessafe.com
 - Modern Dog Magazine - How to Read Body Language -
<http://www.moderndogmagazine.com/articles/how-read-your-dogs-body-language/415>
 - Yin S. Online movie clip and poster - http://drsophiayin.com/resources/video_full/dog-bite-prevention-psa-why-dogs-bite-and-what-to-avoid
 - Zoom Room Guide to Body Language: https://www.youtube.com/watch?v=00_9JPltXHI

CANINE AGGRESSION: DRUGS AND SUPPLEMENTS

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When the dog is excessively aroused, fearful, anxious, overly reactive lacking impulse control or “behaviourally abnormal”, psychotropic medications are indicated to improve the problem as well as address the dog’s well-being. However, drugs do not change the relationship with the stimulus; therefore, concurrent behavior modification is needed to desensitize, countercondition and train desirable.

Selective serotonin reuptake inhibitors might be most effective for hyperactivity, aggression, social anxiety, generalized fear and anxiety and panic disorders. Four weeks or longer is generally required to achieve full therapeutic effects. Starting the medication at the time of the consultation allows time for the drug to reach optimal therapeutic effect when the exposure program begins. Medication might not be required for dogs that can be effectively kept away from fear- evoking situations, provided the dog is sufficiently settled and relaxed. Adjunctive medication to further reduce anxiety especially prior to stimulus exposure might include benzodiazepines, trazodone, clonidine or propranolol, alone or in combination. If effective these drugs might be used several times a day.

Evidence and drug selection

Evidence based decision making allows treatment options to be selected using the available evidence together with the needs of the patient, client and problem. To date there are no randomized placebo controlled trials (RCT) for medications for the treatment of aggression in dogs. Yet in veterinary behaviour the placebo effect can reach 50% or higher.¹ Laboratory models also provide a standardized measure for evaluating therapeutic effect with minimal subject variability and no owner bias.² In veterinary behaviour drug information is often extrapolated from human literature; however, metabolism and effects vary between species and individuals. For example the clearance $\frac{1}{2}$ life of diazepam and its active intermediate metabolite nordiazepam in dogs is 2.5-3 hours and in humans up to 48 hours for diazepam and 100 for nordiazepam. When dose, compliance or availability is an issue compounding is an option; however, stability, storage and bio-availability are concerns.

Psychotropic drugs

Selective serotonin reuptake inhibitors (SSRI) are most commonly used in dogs that are behaviourally abnormal, to control reactivity and impulsivity, reduce fear and anxiety and improve trainability as well as address the dog’s behavioral well-being. SSRI’s are selective in blocking the reuptake of 5HT1A into the presynaptic neurons. Fluoxetine and paroxetine might be useful for general anxiety disorders, stabilizing mood, reducing impulsivity and behaviorally pathologic aggression. Fluoxetine and fluvoxamine might be effective for hyperactivity and aggression; paroxetine for social anxiety and panic disorders; and sertraline may be effective for irritable aggression, generalized fear and anxiety and social aggression.

The primary mechanism of action of TCA’s is to block the reuptake of serotonin and to a lesser extent noradrenaline. They also have anticholinergic and antihistaminic effects which may contribute to varying levels of sedation, urine and stool retention. Clomipramine and

amitriptyline may be useful in controlling underlying anxiety and impulsivity in aggressive dogs. However, studies have shown no effect of amitriptyline or clomipramine on canine aggression.^{3,4}

While antidepressants reach peak plasma levels within hours, reuptake inhibition may induce down-regulation of postsynaptic receptors that are responsible for clinical effects. Therefore, 4 weeks or longer is generally recommended to fully assess therapeutic effects. Starting the medication at the time of the consultation allows time for the drug to reach optimal therapeutic effect when the exposure program begins. There is extensive evidence for the use of clomipramine and fluoxetine for treating generalized anxiety and compulsive disorders and case evidence for the use of SSRI's for social phobias.

Buspirone is a serotonin (5HT_{1A}) receptor agonist and a dopamine (D₂) agonist. It is used for mild fear and anxiety. It is non-sedating, does not stimulate appetite, and does not inhibit memory. It takes a week or more to reach effect. Adding buspirone to an SSRI might help to insure an adequate serotonin pool.

Benzodiazepines potentiate the effects of (GABA), an inhibitory neurotransmitter. They cause a decrease in anxiety, hyperphagia, and muscle relaxation. They have a rapid onset and may have a rebound effect on withdrawal. They can be used alone or adjunctively primarily on an as needed basis but may be considered in select cases on an ongoing basis with multiple daily dosing.^{5,6} They may cause paradoxical excitability, increased activity, and an amnesic effect. They might be useful for drug desensitization and counterconditioning. Buspirone and benzodiazepines can disinhibit which may result in aggression.

Beta blockers such as propranolol reduce physiologic signs of anxiety (heart rate, respiratory rate, trembling). Therefore they might be most useful if combined with drugs that reduce behavioural anxiety.⁵ Clonidine a selective alpha-2 agonist that blocks noradrenaline, might be used together with SSRI's for situational use in fear or territorial aggression, separation anxiety, nocturnal barking, or noise phobias.⁷

Trazodone, a serotonin 2A antagonist-reuptake inhibitor, may be useful in dogs for generalized anxiety, separation anxiety, storm phobias, and some forms of aggression including interdog aggression and impulse control disorders. Trazodone can be used on as needed basis alone or in conjunction with a TCA or SSRI or 2 to 3 times daily.⁸

Focal seizures of the temporal lobe may present with mood alterations or hallucinatory and self-traumatic behaviours. Generalized seizures may be associated with aggression e.g. in the post-ictal phase. Therefore anticonvulsants may be a consideration in diagnosis and treatment. Levetiracetam may be effective for focal seizures, and for anxiety, panic, and mood disorders which may have comorbidity with epilepsy. Gabapentin might be combined with SSRI's for the treatment of impulse control disorders, noise phobias and to reduce reactivity. Carbamazepine is also a mood stabilizer that may be a useful adjunct to SSRI's for irritable and impulsive aggression.

Neuroleptics decrease motor function at the level of the basal ganglia in the brain, elevate prolactin levels and may reduce aggression as dopamine antagonists. Phenothiazines such as acepromazine are sedatives but do not reduce anxiety.

Selegiline is an MAOB inhibitor licensed for CDS in North America, and emotional disorders in Europe. Chronic stress associated with stereotypic and displacement behaviours, fear aggression, and autonomic signs, may have elevated prolactin levels, which might improve with selegiline, while lower prolactin levels are seen with acute onset fears and phobias which might improve with fluoxetine therapy.⁹

Complementary and alternative medications (CAM's) are another option; however, few have been assessed in evidence based studies. Yet these products have appeal because they are considered "natural", are available OTC and have extensive anecdotal support. Products that might be useful in reducing anxiety and improving trainability include Adaptil, alpha-casozepine, l-theanine, melatonin, Harmonease and aromatherapy. Each of these might be used concurrently with drug therapy. Aggression might be reduced by supplementing tryptophan to a reduced protein diet (to optimize entry through the blood brain barrier). In addition, adding tryptophan to an SSRI or TCA may increase the available serotonin pool. Royal Canin Calm diet contains both alpha-casozepine and l-tryptophan. There have been no studies to demonstrate efficacy of other natural products including Bach flower remedies or homeopathy.

Abnormal aggressive dogs

For most cases of behaviourally abnormal dogs an SSRI such as fluoxetine or paroxetine would be the first choice for managing underlying anxiety and impulsivity. Immediate acting medications might be needed concurrently prior to specific events including benzodiazepines (e.g. alprazolam, lorazepam, diazepam), trazodone, clonidine, or propranolol. Natural products might also be used concurrently. In some cases drug combinations will need to be considered such as a combination of SSRI with carbamazepine, gabapentin, clonidine, trazodone, buspirone or even a TCA (with cautious monitoring for serotonin syndrome).

Drug doses for behaviour therapy

	Dose
Alprazolam	0.02-0.1 mg/kg bid to qid
Clonazepam	0.1-1.0 mg/kg bid to prn
Diazepam	0.5-2 mg/kg prn to q6h
Lorazepam	0.25-0.2 mg/kg sid to prn
Amitriptyline	2.0-4.0mg/kg bid
Clomipramine	1-3 mg/kg bid
Citalopram	0.5-2.0 mg/kg sid
Fluoxetine	1.0 – 2.0 mg/kg sid
Fluvoxamine	1.0 -2.0 mg/kg sid – bid
Paroxetine	0.5-2.0 mg/kg sid
Sertraline	1-5 mg/kg sid or divided bid
Clonidine	0.01-0.05mg/kg prn to tid
Propranolol	0.5-3.0 mg/kg bid or prn
Buspirone	0.5-2.0 mg/kg sid-tid

Trazodone	2 to 8 mg/kg prn to tid (up to 15 mg/kg prn)
Gabapentin	10-30 mg/kg bid to tid
Carbamazepine	4-8 mg/kg bid to tid
Levetiracetam	20 mg/kg tid
Selegiline	0.5-1 mg/kg sid in am

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COGNITIVE DYSFUNCTION SYNDROME – DO PETS GET ALZHEIMERS?

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Cognitive dysfunction syndrome (CDS) is a neurodegenerative disorder of senior dogs and cats which is characterized by a gradual cognitive decline and increasing brain pathology. The diagnosis is based on clinical signs described by the acronym DISHA; **D**isorientation; altered social **I**nteractions c) altered **S**leep-wake cycles d) **H**ousesoiling and e) **A**ltered activity levels. An increase in anxiety is also reported.^{1,2}

While the decline in learning and memory may be the most important indicator of cognitive decline, the average pet may appear minimally challenged. Therefore the development and validation of tests for assessing cognitive function in the laboratory (e.g. spatial memory, attention, discrimination, reversal) has been instrumental in identifying age related deficits in learning and memory.^{1,3-5} In fact, dogs and cats may show impairment as early as 6 to 8 years of age. Although impractical for clinical use, similar tasks have been developed that also demonstrate deficits in pet dogs including DNMP and an open field food search. From a clinical perspective, a decrease in performance of previously learned commands, learned behaviors, breed specific activities (retrieve, hunt) or a decline in ability to learn new tasks might be seen.

PREVALENCE

Not all dogs and cats will develop CDS. Prevalence of cognitive impairment from 22.5% to 74% has been reported.^{6,7} In one recent study by Salvin et al., prevalence of CDS in dogs ranged from 5% in dogs 10-12 to 41% in dogs over 14 with an overall prevalence of 14.2%.¹² In a cat study, 35% had signs consistent with CDS; 28% of 95 cats aged 11 to 15 and 50% of 46 cats over 15.⁸

Both prevalence and severity increase with age. In one study of 215 dogs over 6 months 42% of dogs with no impairment progressed to mild impairment and 24% from mild to moderate impairment. This rose to 71.4% converting from none to mild and 50% from mild to moderate after 1 year.⁶ Another study of 94 dogs over 8 years of age found that 58% of dogs with no signs of CDS progressed to borderline cognitive dysfunction, and 11% of dogs moved from borderline to CDS.² In both studies dogs with no impairment did not progress to dementia.

BEHAVIOR SIGNS IN SENIOR PETS

a) Owner reported signs

The prevalence of behavioral signs in senior pets will vary with the caseload. For example the most commonly reported signs in senior pets at behavior referral practices reflect those that are sufficiently problematic to the pet or the owner to seek help. While CDS may be an underlying factor, other neurologic diseases, sensory decline, endocrine and metabolic disorders, musculoskeletal disease and other causes of pain must be ruled out.

In one study of 270 dogs over 7 years of age presented for behavior problems, 32% displayed aggression to family members, 16% aggression to family dogs, 9% barking, 8% separation anxiety, 6.4% disorientation, 6% aggression towards unfamiliar people, 5% housesoiling, 4.2% destructive, 4% compulsive disorders and 3% noise fears.⁸ Of 83 cats referred for behavioral consultations most cats

presented with marking or soiling (73%), followed by aggression (16%), vocalization (6%) and restlessness (6%).¹

b) Cognitive decline and dysfunction

As signs of cognitive decline may initially be subtle and pet owners unaware that treatment options are available, many cases go unreported until the signs become problematic for the owners, or a welfare issue for the pet. However, early detection allows for early intervention so that further decline might be slowed and behavioral signs improved. In one survey owners reported only 12% of pets with signs, while in the Salvin study, 85% of cases had not been diagnosed.⁷ Therefore veterinarians must be proactive in questioning owners as to the presence of signs. Of 957 dogs 8 years and older, when compared to their behavior 6 months previously, more than half of the behaviors showed a greater incidence of deterioration and two thirds showed a significant age related deterioration in severity. Activity and play levels, response to commands, and fears and phobias deteriorated most, although medical causes may have been an underlying cause.¹⁰ In a second study of 94 dogs over 8 years of age, that had been thoroughly screened to rule out medical problems, the most common signs of CDS were sleeping more during the day and restless at night (57%), altered social interactions (51%), disorientation (49%) and anxiety (46%).² For dogs with mild cognitive dysfunction, the predominant sign was daytime sleep (70%) with anxiety in 11% of dogs while anxiety in the non-cognitive dysfunction dogs was 4%. The most commonly reported sign in cats 11-14 was altered social interactions while for cats 15 and over, alterations in activity including aimless activity and vocalization were most common.^{8,3}

DIAGNOSIS OF CDS

When signs of CDS are identified, a diagnostic workup is necessary to rule out medical, physical and motor dysfunction as a cause of the signs. What needs to be assessed and with what diagnostic tools must be based on the behavioral and medical presenting signs, physical examination, and results of baseline screening. Next to neurological disease, sensory decline, endocrine and metabolic disorders and musculoskeletal disease are the primary rule-outs.

AGING AND ITS EFFECT ON THE BRAIN

In dogs, with increasing age frontal lobe volume decreases, ventricular size increases and there is meningeal calcification, demyelination, a reduction in neurons and an increase in toxic free radicals.^{5,11} In cats, there is also neuron loss, increased ventricular size, cerebral atrophy and widening of sulci although not as marked as dogs.⁸ Circulatory changes in dogs and cats including microhemorrhage and infarcts may also be responsible for signs of CDS. A decline in the cholinergic system has also been identified which may contribute to declining cognitive and motor function.¹² In dogs, cats and humans there is an accumulation of diffuse beta amyloid plaques and perivascular infiltrates^{11,13,14} Increased A β is positively correlated with cognitive impairment in dogs but results in cats are variable and numbers are low.^{8,14} The most striking difference from humans is the absence of neurofibrillary tangles in dogs and cats although hyperphosphorylated tau is reported. Most recently cognitive decline has been shown to be related to neuroinflammation and tau hyperphosphorylation in synapses in dogs.¹⁵

ENVIRONMENTAL MANAGEMENT AND COGNITIVE ENRICHMENT

When cognition is impaired, diet, drugs or supplements might be useful in improving signs and slowing the progress of CDS. Canine studies have demonstrated that mental stimulation in the form of training, play, exercise and manipulation toys can help to maintain quality of life as well as cognitive function.¹⁵

MEDICAL THERAPY

Selegiline is an inhibitor of monoamine oxidase B in the dog which has demonstrated efficacy in improving cognitive signs. It has been shown to increase 2-phenylethylamine in the dog brain, a neuromodulator that enhances dopamine and other catecholamines in the cortex and hippocampus. It may also contribute to a decrease in free radical load through decreased production and increased clearance. Dose is 0.5 – 1.0 mg/kg daily.

Since the elderly are particularly susceptible to the effects of anticholinergic drugs, it is prudent to avoid drugs with anticholinergic effects.¹⁶ In fact, drugs or natural products that enhance cholinergic transmission might have potential benefits for improving signs of CDS in dogs and cats.¹² Propentofylline, a xanthine derivative is licensed for lethargy and depressed demeanour in old dogs in some countries but not in North America. It may increase blood flow and inhibit platelet aggregation and thrombus formation. Other treatment strategies include nicergoline an alpha 1 and alpha 2 agonist and the NMDA antagonist memantine. No drugs are approved for cats; however, selegiline and propentofylline may be useful. .

A primary therapeutic strategy for cognitive dysfunction in dogs, cats and humans is to reduce the risk factors that contribute to cognitive decline. It is likely that an integrative approach is required such as a diet fortified with antioxidants and polyunsaturated fatty acids. In dogs, a senior diet (Canine b/d, Hills Pet Nutrition) has been shown to improve signs and slow the progress of cognitive decline. It is supplemented with fatty acids, antioxidants (vitamins C and E, beta carotene, selenium, flavonoids, carotenoids), and dl-alpha-lipoic diet and l-carnitine to enhance mitochondrial function.^{16,17} The combined effect of the diet plus an enriched environment provided the greatest improvement.¹⁶ However, while enrichment resulted in improvement in cognitive function, the dietary therapy resulted in a reduction in reactive oxygen species and in beta-amyloid accumulation.¹⁷

A Purina Veterinary Diet (Essential Care Senior, Pro Plan Bright Minds), supplemented with botanic oils containing medium chain triglycerides to provide ketone bodies as an alternate source of energy for aging neurons, has also been shown to significantly improve CDS in dogs.¹⁸ For cats, a diet (not yet commercially available) developed by Nestle Purina supplemented with antioxidants (Vitamins E and C and Selenium), arginine, B vitamins and fish improved learning and memory tasks compared to a control diet in cats 5.5-8.7 years.³

Senilife® (CEVA Animal Health), has demonstrated efficacy in improving cognition in both a laboratory model and clinical studies in dogs. It contains phosphatidylserine, a membrane phospholipid as well as Gingko biloba, vitamins E and B6 and resveratrol. Another product with phosphatidylserine, omega-3 fatty acids, vitamins E and C, l-carnitine, alpha-lipoic acid, coenzyme Q and selenium his available in the UK. The cat product has no alpha-lipoic acid.

S-adenosyl-l-methionine (Novifit®, Virbac) may help to maintain cell membrane fluidity and receptor function, regulate neurotransmitter levels and increase production of glutathione. Improvement has been demonstrated in dogs in a placebo controlled trial and in laboratory studies in both dogs and cats.

Apoaequorin (Neutricks™) is a protein found in jellyfish that in laboratory trials improved learning and attention in dogs. It is a calcium buffering protein that may provide neuroprotection against aging.

BEHAVIOR MODIFICATION AND ADJUNCTIVE MEDICATIONS

Together with the treatment for CDS, psychotropic medications may be required to manage underlying stress and address those signs such as night waking, agitation and anxiety that continue to be problematic for the owner and pet. In addition, clinical signs may persist even if CDS and underlying anxiety and stress have been effectively addressed. Therefore concurrent environmental management and behavior modification are also likely to be needed.

Since anticholinergic drugs should be avoided, SSRI's or buspirone might be preferred options for ongoing use. Trazodone might also be considered either alone or in combination with an ongoing SSRI or buspirone. However, drugs that increase serotonin, should not be used concurrently with selegiline. While benzodiazepines could contribute to further cognitive deficits, they may be useful in managing signs of anxiety and sleep disturbances. Lorazepam, clonazepam, and oxazepam are preferred since they have no active intermediate metabolites. Adjunctive use of propranolol or clonidine may reduce some of the noradrenergic effects contributing to the signs of anxiety. Gabapentin might reduce reactivity and neuropathic pain. Natural products might also aid in the control of anxiety.

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How to Help Dogs With Sound Sensitivities From a Training Perspective
Mikkel Becker, CPDT

Tone: Warm, informative, confident, honest.

Message: How to prevent sound sensitivities. If a dog has sensitivity, how to manage it and strategies to change the animal's perception to situations where they may be afraid.

Focus: Strategies for prevention, early treatment and treatment in progressed phobia with noise.

50 minutes

- A. Inborn tendency for fear:
 - 1. Study published Applied Animal Behavior Science. Sensitivity owner survey of 5,257 dogs from 17 different breeds. Thunderstorms, heavy traffic, fireworks, and loud noises (banging sounds/gunshots).
 - a) 23% scored a 5 (highest, very strong anxiety) for at least one of the four noises and deemed noise sensitive.
 - b) Fireworks evoked strongest reaction. Situations with loud noises/gunshots, thunderstorms and heavy traffic following in decreasing order.
 - c) Fear responses to loud noises such as fireworks, gunshots and thunder commonly co-occur- generalization between salient stimuli.
 - d) Origin- dogs living with the owner who bred them had a reduced risk compared to dogs purchased from the breeder by a second owner. Early environment being similar to environment in adulthood is protective.
 - e) Older dogs were more anxious
 - f) Females were 30 percent more sensitive to noise than males.
 - (1) Genetic differences in males and females.
 - (2) Or, hormonal. Neutered animals 72 % more likely to cower in fear than un-neutered.
 - g) Breed differences
 - (1) More sensitive: Norwegian Buhunds, Irish Coated Wheaten Terrier, Lagotta Romagnolo.
 - (2) Least affected: Great Dane, Chinese Crested, Boxer.
 - h) Sound sensitive dogs at higher risk of suffering other effects of fear.
 - (1) Three times more likely to have separation anxiety.
 - (2) 18 times more likely to show signs of being afraid in novel situations.
 - (3) 4 times more likely to take longer to calm down after stressful situation. Took longer to calm down after stress inducing situation.
 - (4) Noise sensitivity may be indicator of an underlying physiological mechanism causing dogs to be more reactive to stressors.
 - (5) Noise sensitivity could be measure of a dog's likelihood to display fear in other situations.
- B. Early learning
 - 1. Animals still have genetic components increasing chance of fear. But, can have some positive experience in the bank account.

2. Tackling fear early when puppy has more bounce back is easier than tackling it later when the brain is less flexible.
 3. Puppy Socialization:
 - i) Socialization
 - ii) What does a puppy experience or not experience?
 - iii) Habituation or sensitization?
 - iv) What was their experience like?
 - v) Many owners don't recognize fear.
 - vi) If owner does recognize it, may not address it.
 - vii) Study:
 4. Development: Dogs mature 9 months-2 years especially marked reactions.
 5. Older dogs may have had it, but less bounce back later.
- C. Trigger combined with genetic tendency leaving dog more vulnerable.
- (a) Prevention: Socializing not just the sights of feelings of experience, the sounds as well.
 2. Scary Experience (4th of July, garbage day, car backfiring).
 3. Maturing dog (second fear period, less socialization as grow, reaching sexual maturity fear may be more evident- 9 months to two years).
 4. Brain less able to bounce back from stressor.
 - a) Each fearful experience can ingrain patterns of fear in the brain.
 5. Phobic reaction. One time event.
 - a) Triggers set up animal to full blown fear reaction. Related to factors of original situation
 - (a) Beeping, alarm, reboot noise,
 - b) Fear with other sounds with similar qualities.
 - (a) Generalizes- thunder to traffic, guns, fireworks, car backfiring, washer/dryer, garbage day, rumble strips, sounds on television, lawn mower, tags on the dish.
- D. Prevention:
1. Recognizing fear and addressing early.
 - a) Half of owners reported at least one behavioral sign indicative of fear. But only 1/4 of owners reported their dog as fearful of noises.
 - (1) Most common: Vocalizing, trembling/shaking, hiding, seeking people.
 - (2) Urination, salivation, destruction may cause anger or disappointment in humans and may affect their interpretation of fear.
 2. Body language of fear and tension or relaxed and happy dog (dependent upon individual and some breed related tendencies).
 - a) Hiding (under bed, next to toilet)
 - b) Panting
 - c) Pacing
 - d) Tremble/Shiver/Shake
 - e) Salivating/drooling
 - f) Whining
 - g) Barking
 - h) Escape (digging, pawing, scratching, chewing at exit points)
 - i) Running away

- j) Urination
 - k) Defecation
 - l) Dilated pupils
 - m) Inability to settle for long
 - n) Shut down
 - o) Excessive shadowing of person
 - p) Reluctance to eat
 - q) Reluctance to listen to known cues/commands
3. Less than 1/3 owners sought professional advice for treatment of their pet's fear and response to noise.
 4. Can become dangerous: Escape behaviors (potential for harm), running away, chronic stress leading to immune compromise and premature death.
 5. Addressing on puppy visits. First visits. Continuing question. If note any alerting to noise.
 6. Paying attention to the early environment the animal was in, including sound exposure. Early environment influences comfort of animal in adult living environment.
 - a) Protective quality of the animal raised around sound environment. Habituation.
 - b) Rescue/foster/breeder- important to consider what animal was exposed to.
 - c) In utero babies can become conditioned to noises. condition dogs to this as well (link to study?)
 7. Sound exposure socialization
 - a) Purposed field trips to socialize to sights/ feelings/ sounds!
 - b) If animal fearful, teaching how to work with fear. Letting animal discover it on their own. Giving direction.
 - c) Socialization sounds: Play during meals/play/training.
 - (1) Baby cry. Young children
 - (2) Thunder
 - (3) Fireworks
 - (4) Traffic/ trucks/ motorcycles
 - (5) Gun fire
 - (6) Animal noises
 - (7) Dogs barking
 - (8) Lawn mower
- E. Trainer's how to help dogs with sound sensitivities.
1. Changing association with noises (not always possible, may need other calming strategies explained below).
 - a) Start at very low exposure/ volume if possible
 - b) Level animal notices, but isn't upset by.
 - c) If animal becomes upset, lower and get a more relaxed response before stopping.
 - d) Just exposure may not help. Could worsen. Flooding can sensitize.
 - (1) Thunderstorms
 - (2) July fourth
 - (3) Avoiding altogether if possible or using medication important.
 - e) Vacuum in other room. Door closed. Then door open. Movement separate.
 - f) Starting away from traffic not on the busy road if dog is fearful.
 - (1) Sensitizing a dog by flooding.

2. Training exercises to combine with gradual exposure.
 - a) Mealtime or training sessions around sounds (recordings)
 - (1) Snickers with sirens
 - (2) Bear with traffic and construction.
 - (3) Traffic noise (reward simply for hearing it).
 - (4) Garbage day
 - (5) Clickers in class/ heater turning on/ bird squawking
 - (6) Other dogs. Fear related. Addressing sounds causing alarm. CC.
 - (a) Treat when other dog barks and your dog quiet. Dogs soon look to their person.
 - (b) Jingling tags. Walking dog.
 - (c) Not taking treats is sign it's 'too much'.
 - (d) May start with 'listen' then later move to asking to do something like 'heel'.
 - b) 'Hear that?' game.
 - (1) Puppy class. Adult dogs.
 - (2) QT with traffic.
 - (3) Click looking over at the noise then eventually full circle to turning back.
 - (4) Automatic check in reward.
 - (5) Give dog something else to do/ focus on.
 - (6) Dogs find their person with this game (VS video link. Works for barking and fear issues).
 - (7) Rewarding relaxation/ confidence.
 - (a) Ears forward
 - (b) Tail more neutral/relaxed
 - (c) Muscle relax
 - (d) Check in (rather than focus on 'scary thing').
 - c) Rewarding confidence
 - (1) Ears forward
 - (2) Tail neutral/relaxing
 - (3) Muscle relax
 - (4) Check in (focusing on you rather than 'scary thing')
 - (5) Rewarding resting on bed, engaging with toy.
 - d) Outside protocol
 - (1) Training walks.
 - (2) Avoiding trigger areas.
 - (3) At first sign of alert, be ready.
 - (4) Listen game.
 - (5) Reward automatic check in's (practice with other things besides noises: new people, sights).
 - (6) Alert at cue- ask to do something (look, touch, let's go!).
 - (7) Toy crazy dogs (have tug/ball on rope/chase toy).
 - (8) Condition to being picked up.
 - (9) Proper equipment: Double leash? Limited slip?
3. Calming strategies:
 - a) Create Puppy Palace (safe haven)

- (1) Condition dog to go there on cue
 - (2) Conditions when dog retreats to be like training scenario. Scent, sights, feel to cue comfort.
 - (3) Cue what the dog already does (bunker/ foxhole).
 - (4) Relaxing on mat/bed/blanket
 - (5) Animal's already chosen spot, closet, basement, bathroom, bathtub, crate with door open, dog house with door open.
 - (6) Covered crate/house or room where outside noise doesn't carry easily.
 - (7) Setup the environment
 - (a) Buffer noise: White noise, music, TV, blankets
 - (b) Buffer visual if needed (close blinds for flashing lights of fireworks or lightning).
 - (c) Comfort items (Scents of Security, Stuffed dog, snuggling blankets, hiding areas underneath, chews/ food puzzles, destuffing toys)
 - (d) Static buildup- dryer sheet/tile
 - (e) Relaxation cues (cue word/ lavender or chamomile scent or owner scent/ pressure garment).
- b) Sound protection
- (1) Can't eliminate sounds, so let's use noise to our advantage.
 - (2) Calming music. Masks outside or upsetting noise
 - (a) TV or radio not enough. Type of music influences the animal.
 - (b) Classical/species specific music:
 - i) Beat of resting heart rate.
 - ii) Classical music increases sleep, resting postures, decreased barking.
 - iii) Heavy metal increases body shaking, standing.
 - iv) Longer notes, loops/repeating soothes and calms.
 - v) Short, rapid repetition increase motor activity.
 - vi) No change in shelter dogs listening to pop, no music or human voice.
 - (c) Recommendations:
 - i) Classical
 - ii) Through a Dog's Ear (driving edition available)
 - iii) iCalm Pet
 - iv) Pet Acoustics (collar/ portable).
 - v) Laughing dogs soundtrack
 - (3) Noise reduction
 - (a) Mutt muffs
 - (b) Happy Hoodie
 - (c) Sound proofing: Blankets, insulation (sound studio).
 - (d) White noise machine (app, types, fountain/fan). May use in combination for lulls in music.
 - (4) Addressing noise with multiple animals.
 - (a) Shelter/hospital other vocalizations.
 - (b) Animals can interpret other animal's vocalizations and emotional state contained within.
 - (c) Noise pollution and stress/sleep.
 - i) Creating a space with sound in mind.

- ii) Music. White noise.
 - iii) Quiet times. Nap.
 - iv) Organizing to make times going in and out minimal.
 - v) Eliminating triggers as possible (blocking visual).
 - vi) Training for quiet (reward quiet).
- c) Changes for comfort
 - (1) Enrichment
 - (a) Food puzzles
 - (b) Thinking games
 - (c) Walks
 - (d) Socialization
 - (e) Play
 - (2) Activity
 - (a) Moving to release stress
 - (b) Run with me game (switch between run/walk).
 - (c) Chase toy (flirt pole, stuffed toy on rope).
 - (d) Come when called between people.
 - (e) Find it.
 - (f) Treasure hunt
 - (g) Homemade agility course
 - (3) Cue behavior likely to elicit a response
 - (a) Howl
 - (b) Favorite treat (Cue: Treat! Dingo! Turkey! Bacon!)
 - i) Audition treats to have the highest value available.
 - (4) Tactile
 - (a) Massage, T-touch.
 - (b) Petting
 - i) Myth of 'don't comfort'.
 - ii) Doesn't decrease or elevate cortisol.
 - iii) In other situations cortisol doesn't decrease for a dog when they're pet as it does for people.
 - iv) Other benefits of petting:
 - (1) hormones and neurotransmitters like oxytocin, prolactin and beta-endorphin increased (associated with good feelings and bonding).
 - v) If animal is worsening, absolutely don't do it. Watch how they respond.
 - vi) If petting dog and they are being reinforced, likely reinforced for being near you rather than other behaviors like scratching exit points, pacing, vocalizing.
 - vii) Study tested duration dogs liked. Stayed there full 18 minutes it was offered.
 - (c) Compression garment
 - (5) Another animal (greatest effect on lowering cortisol).
 - (a) Calm animal
 - (6) Scents

- (a) Lavender/ chamomile (scent cue to relax).
- (7) Puppy Pacifier
 - (a) Stuffed food puzzle (freezing)
 - (b) Puzzle needed moved actively
 - (c) Toy stuffed with multiple items
 - (d) Chew
- (8) Comfort item
 - (a) Snuggle puppy
 - (b) Scents of security
 - (c) Item of their person
- (9) Medication/ Supplements
 - (a) Adaptil (collar/diffuser/spray).
 - (b) Zylkene
 - (c) Anxitane
 - (d) Melatonin
 - (e) CALM diet.
 - (f) SSRI's. Short acting.
- (10) Interaction with the pet (training above).
 - (a) Some animals best left alone.
 - (b) Many will seek attention (one of the signs of anxiety).
 - (c) If animal is hiding though, don't attempt to pull the dog out physically from hiding space.
 - (d) Some dogs may seek out their people or be comforted by presence of person nearby.
 - (e) Study shows petting more appreciated than talking.
 - (f) No more interest in human praise than having no interaction at all.
 - (g) No difference with petting.
 - i) Conditioned words- cue words like walk, or cued words with positive association.
 - ii) Marker words (good girl, good dog, yes, good).
 - (h) Emotional contagion- spread of fear within a group. Animals pick up on our cues of fear or relaxation. How we react they respond to (cat with fan, find dog study).
 - i) Teaching pet owners to calm themselves (my article resource)
 - ii) Pet professionals calming ourselves (my article resource).

F. Addressing related fears.

- 1. Separation
 - a) Safeguards in place when pet left alone
 - b) Pet may be less resistant to rise when left alone... combination of anxiety when alone, less security without family present.
 - c) Safeguards in place for certain days (storm/ garbage day).
 - d) Separation exercises
 - (1) Independence
 - (2) Mat training
 - (3) Relaxed leaving and return.
 - (4) Change cues

- (5) Pay attention to dog when calm
 - (6) Short leaving and coming back. Special cue.
- 2. Building confidence and 'bounce back'.
 - a) Minimally stressful experiences early on
 - b) Teaching person how to recognize fear early
 - c) Teaching coping techniques to the dog.
- G. Noises in the car.
 - 1. Music. Classical Music. Driving edition species music.
 - 2. Safe space in car (crate).
 - 3. Pay attention to talk.
 - 4. Rumble strips
 - 5. Freeway vs. neighborhood
 - 6. Distraction/calming items
- H. Veterinary hospital noises
 - 1. Species music for rooms.
 - 2. iCalm Dog.
 - 3. Sound muffled
 - 4. Lower alert noises if possible (lower volume, light?) Phone, door.
 - 5. Separate entrances/rooms for fearful animals.
 - a) Reactive animals (ID tags, nails walking).
 - 6. Treatment area/recovery area separate
 - 7. Species specific areas in hospital
 - a) Parrot
 - b) Dogs barking
 - c) Other animals communication understood and may be reacted upon.
 - 8. Close crates/ doors softly

How to be a Behavior and Training Friendly Clinic Through Client Education.
Mikkel Becker, CPDT

Subject

- I. Why focus on behavior and training:
 - A. Animal's emotional wellbeing is a commitment of the Fear Free model (keeps pets, humans safe in clinic and home).
 1. Increasing client compliance with visits
 2. Increasing client loyalty to clinic
 3. Ultimately keeping people and animals safe during visits.
 4. Training and behavior is a major need. One of the most searched terms on the internet (find stat).
 - B. Physical, mental, emotional wellbeing of the pet overseen and cared for throughout their lifetime
 1. Behavior issues are a main reason for surrender and euthanasia.
 2. Changes in behavior need carefully watched to address problems early
 - a) May point to a health problem
 - b) Addressing aggression early
 - c) Young pets are most at risk for early surrender
 3. Cats are particularly in need of behavior help
 - a) Trainability of cats is not understood.
 - (1) People know to socialize a puppy, but what about the kitten?
 - (2) Behavior issues may not be resolved
 - (a) People often 'live with it' or get rid of the cat
 - i) That's just how he is.
 - (3) Being seen as a resource lets cats get all of their particular needs met to resolve an issue
 - (a) Medical issue? Stress? Learned?
 - b) Elimination outside of the litter box
 - c) Environmental help in the home
 - d) Teaching people behavior help is available Selection of the right pet
 4. As a behavior resource, particularly relevant in choosing a new pet
 - a) Is a pet right for the family?
 - (1) Other animals already in the home.
 - (a) Is another animal a good fit?
 - (b) What animal is the right fit?
 - i) Age
 - ii) Breed/Size
 - iii) Temperament
 - iv) Health concerns
 - v) Family life
 - (1) What is likely future?
 - (a) Kids?
 - (b) Grandkids?

- (c) Move?
 - (d) Career/work commitment?
 - (2) Resources for adoption
 - (a) Rescue groups
 - (b) Reputable breeders
 - (3) Setup right
 - b) What qualities do we look for?
 - c) Looking to the future
 - (1) Family life
 - (2) Other pets
 - (3) Lifestyle (city/country)
- 5. Medical oversight according to changes in behavior.
 - a) More people are interested in spay/neuter according to behavior
 - b) Puppyhood and adolescent changes to reaching sexual maturity and senior years.
- 6. Medications and supplements as needed with differing behavior issues.
 - a) Sound sensitivity
 - b) Compulsive behavior
 - c) Anxiety, phobia, specific fears
 - d) Aggression

II. Where to start

A. Start where you are and build

- 1. Fitting into regular visit
 - a) Phone/email setup prior (Fear Free instructions could also briefly monitor and flag problems).
 - b) Fill out while they wait
 - c) Technician to ask important behavior questions with vet to followup during medical review.
 - d) May need followup visit or direct referral to resources.
- 2. Adjusting routine for pet's behavior
 - a) Followup visit
 - (1) Rather than forging through nail trim, suggest coming back with another approach.
 - b) Schedule in longer visit
 - (1) Low traffic times/ Extra hands
 - (2) Technician with behavior expertise
 - (3) Charge extra
- 3. Helping with followup care and visits
 - a) Medications
 - b) Procedures

B. Resources

- 1. Trainers
 - a) Not a separate entity.
 - b) Methods need monitored.
 - (1) What type of training?
 - (2) Risks of force based training

- (3) Certification?
 - c) Trying it out yourself
 - d) Many have specialities
 - 2. Behavior technicians
 - a) Certification
 - b) Specialty
 - c) Already have someone skilled?
 - 3. (1) Clinics and learning opportunities
 - Behavior professionals
 - a) Other certifications to look for
 - 4. Veterinary behaviorist
 - a) Referral
 - b) Continuing oversight
 - c) Consulting possibilities
 - d) Outreach from behavior resources
 - (1) Purdue
 - e) Partnership between behaviorist, vet, trainer, owner.
- III. Addressing problems
 - A. Exam (#addressed, #followed up on and #resolved)
 - 1. Proving information based upon age/time with family.
 - a) Puppy prevention/problems
 - b) Adolescent dog
 - c) Senior dog
 - d) Breed tendency
 - (1) Higher energy cat or dog breed
 - (2) Herding
 - e) Size issues
 - (1) Small dog
 - 2. Problems to Scout for:
 - a) Puppy/ Kitten
 - b) Adolescent
 - c) Rescue
 - d) Adult going into senior years
 - 3. Noting behavior
 - a) Talking about emotional/behavioral wellbeing
 - (1) Fear Free
 - (2) Dog and cat body language
 - (3) Why it's important to keep stress in check
 - (4) Preventive
 - (5) Addressing problems
 - b) Noting problems and addressing with help or resources
 - (1) Watching behavior (fearful? Aggressive?)
 - (a) Taking note of happy dog and cat body language (I did puppy instead of just fearful dogs).
 - (2) If they mention something like cat spraying, delving into it or pointing towards help.

- c) Followup
 - (1) Referral
 - (2) Training going?
 - (3) Resources for issues
 - 4. Problems with rescue
 - B. Referral
 - C. Working with another professional on an issue
 - 1. Technician, trainer, veterinary behaviorist, other specialist.
 - 2. Taking in observation
 - 3. Letting behavior person know what to look for
 - a) Medications
 - b) Is it improving? Being a resource
 - 4. Following up
 - D. Great resource books (links)
 - E. Followup with trainer.
 - F. Culture of communication
 - 1. Reach out with early observation/concern
- IV. Preventive and ongoing socialization through class
 - A. Overview: Puppy, Fearless Vet Visits, Specialty- kitten, adolescent, AKC Star Puppy, manners, CGC, Cautious Canine
 - B. Puppy
 - 1. Statement
 - 2. Puppy playgroups
 - a) Socializing to hospital, other pups, people
 - b) Matching puppy personality
 - c) Short: 15-30 minutes
 - 3. Puppy class
 - a) Clean area
 - b) Guest appearances
 - c) Puppy play
 - d) Scale
 - e) Exam table
 - f) Tools: stethoscope, temperature,
 - g) Practice procedures: pilling, ear, eye, restraint, collar grab, nail trims
 - h) Walking equipment
 - i) Play
 - j) Resource guarding
 - k) Noise training
 - l) Training
 - (1) Drop it/take it
 - (2) Sit/Down
 - (3) Walking on a loose leash
 - (4) How to greet
 - (5) Potty training
 - (6) 'Pet' cue
 - (7) Target to meet people

- (8) Gentle (treats, skin)
- (9) Crate training
- (10) How to approach something 'scary'
- 4. Field trip (class/ individual/ Puppy and adult)
 - a) Class field trip
 - b) Conducting 'fun visits'
 - (1) When to do it
 - (2) Where to do it
 - (3) What to offer
- C. Cat offerings
 - 1. Cat College
 - a) Body language
 - b) Training
 - c) Home environment
 - d) Rescue/new cat
 - (1) Lifestyle
 - (a) Cats need interaction
 - (b) Preparing pet for leaving
 - i) Traveling
 - (c) Play
 - (2) Home environment
 - (a) Hiding spaces
 - (b) Climbing spaces
 - (c) Children structure
 - (d) Other animals
 - i) Cats
 - ii) Dogs
 - (3) How to address issues
 - (a) Avoid punishment
 - (b) How to train and reward
 - (c) Management
 - (4) Litter box
 - (5) Handling/Medication
 - (6) Crate
 - (7) Separation
 - e) Fear Free
 - (1) Crate
 - (2) Car
 - (3) Handling
 - (4) Pilling
 - (5) Nail trims
 - f) Training resources
 - (1) Addressing in consult: additional resources; behavior/training help, information in special class and links/handouts.

- (2) Litter box, play aggression, tension between cats, adding a new animal to the home, preparing for baby, taking cat home from the vet, pilling, nail trims, handling, petting aggression, separation training.
- 2. Kitten Class
 - a) Why is kitten socialization important?
 - b) Details
 - (1) Frequency
 - (2) Who is it open to?
 - (3) Price
 - c) Covered in class
 - (1) Crate
 - (2) Litter box training
 - (3) Training
 - (4) Harness
 - (5) Socialization
 - (6) How to play with a cat
 - (7) Setting up the home for success
- D. Other offerings
 - 1. Training basics
 - a) Basic learning theory: Rewarding desired behavior, ignoring or removing reward for unwanted behavior.
 - (1) Ignore jumping
 - (2) Stop petting if jump up
 - b) Being specific to the animal
 - (1) Clicker
 - (2) Marker word
 - c) Effective rewards
 - (1) Treats
 - (2) Food
 - (3) Access to a place
 - (4) Access to a desired activity/interaction
 - d) Risk of force
 - 2. Advanced puppy. AKC Star Puppy.
 - 3. Adolescent Dog
 - 4. Manners
 - a) Class
 - (1) Go to spot/ mat or bed
 - (2) Puppy pushups
 - (3) Hold
 - (4) Touch
 - (5) Drop it/ take it
 - (6) On/off
 - b) Canine Good Citizen (link)
 - 5. Fear Free- Fearless Vet Visits
 - a) Hold
 - b) Mat

- c) Puppy pushups
- d) Target
- e) Offer: Paw, bottom, DS temperature
- f) Muzzle training
- g) Fun visits practice aspects of visit
 - (1) Scale
 - (2) Exam
 - (3) Waiting area (mat)
 - (4) Saying hi to staff
 - (5) Receptionist taking into room
 - (6) Handling
 - (7) Look for handout on things to do during the visit
 - (8) Groomer preparation
- 6. Cautious Canine to Confident Canine
- 7. Feisty Fido
- 8. Special learning opportunity
 - a) Preparing for baby
 - b) Children and dogs
 - c) Come when called
 - d) Walking on a loose leash
 - e) Calm greetings
- V. Environment/ Enrichment Help
 - A. Exercise
 - 1. Outings/walks
 - 2. Play/ Doggy sports
 - a) Recommendations; scenting, agility
 - 3. Dog park?
 - 4. Exercise limited
 - a) Other games and indoor exercise: offer link
 - b) Alternative exercise
 - (1) Treadmill (training)
 - (2) Doggy daycare
 - (3) Dog walker
 - B. Enrichment
 - 1. Food
 - a) Food puzzle
 - b) Scavenger hunt
 - 2. Living space
 - a) Climbing areas/hiding areas
 - 3. Things to do
 - a) Thinking toys
 - b) Interactive toys
 - c) Solo play toys
 - C. Types of products to assist in behavior
 - 1. Front clip harness
 - 2. Head halter

3. Standard leash/ waist leash
4. Treat bag
5. Clicker
6. Cat/kitten harness and leash
7. Cat carrier
8. Litter box
9. Litter
10. Treats

Dynamics Between Multiple Animals in the Home

Tips for Settling Conflict Between Dog and Cat

Mikkel Becker, CPDT

Tone: Warm, informative, confident.

Message: Common types of conflict between dogs and dogs/cats. How to recognize a problem.
Changing influencing factors: environmental/setting events, management, behavior modification.

Focus: Helping the pet owner resolve conflict in their household with their dog and another animal (dog/cat).

- I. Dynamics between animals.
 - A. Defining 'normal'
 1. Disputes and conflict resolution are a part of relationships
 2. Most disputes are done with displays and body language averting an actual physical confrontation.
 3. Few dogs desire to do physical harm to one another
 - a) May sound like he wants to 'kill him', but that likelihood is low.
 - (1) Wants distance. Wants to protect his chew toy.
 - b) Fights can sound quite scary. Often the really loud fights are less harmful.
 - c) Most end in 'spit bites'.
 - B. Dogs have certain rules of conduct.
 1. If it's in one dog's possession, there's respectful distance keeping.
 - a) If another dog disrespects this or seemingly is threatening the dog's possession, warnings are likely.
 - b) Certain dogs are more disposed to resource guarding type behavior.
 - c) For the most part, dogs do a good job of handling these conflicts.
 - d) Video of dog conflict
- II. Genetic and early environment blueprint
 - A. Genetics: Parent's fear and sociability influences the puppy or kitten.
 1. If parents are fearful, their offspring are more likely to be fearful.
 2. Evidence stress can transfer (in utero, early life).
 - a) Enough food?
 - b) How does mother react to things?
 - c) Some low level of stress builds resiliency
 - (1) Sound sensitive dogs often take longer to recover from a stressful situation. Teaching stress recovery important.
 3. Breeding.
 - a) Sociability not always accounted for in breeding.
 - (1) Color/size/breed
 - (a) Cocker spaniels
 - (b) Overbreeding
 - (c) Blue pit bulls/chocolate lab

- (d) Backyard breeding without careful safeguard of animal's behavior
 - i) Aggression inherited
 - (1) Show with two aggressive Sharpei's. Happy end with puppies?!
4. Inborn traits
 - a) Personality
 - b) Breed
 - (1) Born to be fierce/ protective/ tenacious?
 - (2) Sociability to work with other dogs?
 - (3) Certain breeds use tendencies in play/interaction
 - (a) Herding
 - (b) Heeling
 - (c) Rush
 - (d) Stalk
 - (e) Alerting
 - (f) Referee
 - (4) Bond more with person, other animals?
- III. Socialization and changes as animal ages
- A. Puppyhood and kitten-hood learn to interact
 - 1. Conflict resolution/play/being gentle
 - a) Claws and teeth hurt
 - B. Learning boundaries
 - 1. Giving space if another asks for space.
 - 2. Using their own body language to ask for space.
 - 3. Using cues to signal it's all in the name of play.
 - C. Conflict Resolution
 - 1. Conflict and resolution are a normal part of interactions.
 - a) Video of animals resolving a conflict. Body language. Movement. Space to resolve without escalation.
 - 2. Conflict as a teacher of boundaries (puppy class).
 - 3. Not a one-sided relationship
 - a) Learn other animals have preferences and boundary lines.
 - b) Learning to slow down play. Be gentle. Heed warnings.
 - c) Using their own body language.
 - (1) What works to get the other dog to give space?
 - (2) Want them to learn little signals, but not to the point of needing to escalate.
 - (3) 'tough it out' might end in a dog who escalates faster and has little enjoyment with other dogs.
 - d) Watching puppy play and keeping it at a low enough arousal level.
 - (1) Some puppy play classes can get a little too amped up.
 - (a) Taking breaks.
 - i) Training breaks
 - ii) Three second rule
 - iii) Long line or collar grab (taught).
 - (b) Different dogs (not always the crazy ones)

- D. Socialization period. 3-7 weeks cats, 3-12 weeks pups.
1. Learn play/ communication
 2. Learn friend and foe
 - a) Cats with rats.
 - b) Dogs with chickens.
 - c) Single puppy or single kitten
 - (1) Chewie
 - (2) Tufts cat
 - (3) Foster cat
 3. Social experience problems
 - a) Adopted too early
 - (1) Bear
 - (2) Lack social experience
 - (3) Depending upon the environment when to adopt. 7-12 week taken from litter.
 - b) Many animals not taken early enough
 - (1) ASVAB
 - c) Lack ongoing socialization
 - (1) After puppy class it's not emphasized as much
 - d) Medical separation
 - (1) QT
 - (2) Medical separation or problem puppyhood to adulthood
 - (a) Pitbull who had surgery in adolescence. Reactive to other dogs.
 - (b) Early separation has pitfalls
 - i) Don't learn from litter
 - ii) Bear
 - (c) *ASVAB statement socialization.*
 - i) Aggression and problems without puppy class that's protective
 - ii) Frenchie who missed play
 - e) Socialization gone wrong
 - (1) Try to 'fix' problem or socialize animal
 - (a) Get another dog/dog park
 4. Ongoing experience and lifetime learning
 - a) Socialization limited
 - (1) After puppy class, experience limited
 - (2) Socialization may wane off and dog enters second fear period
 - b) Stop after a bad experience
 - (1) Dog reacts or has a bad experience, so interaction ended.
 - (2) Animals readily remember the negative (survival response).
 - c) Commonly discontinue walks if dog reacting
 - (1) Frustration escalates
 - (2) When they see infrequent animals, anxiety and agitation builds up
 - (3) Punishment and owner reaction can escalate reaction
 - d) Experience and change overtime
 - (1) Negative experience.
 - (a) Bruce attacked.

- (b) Dog attacked by off leash dog (fb)
- (2) Lack of positive experience.
 - (a) Heelers.
- (3) Learned from another dog (study learn from observation)
 - (a) Nelson
- (4) Changes overtime
 - (a) Adolescence
 - i) Dogs less tolerant
 - (b) Sexual maturity
 - i) Young male dogs may respond differently towards.
 - (c) Maturing dog
 - i) As dog matures more selective on friendships
 - ii) Desire to play and tolerance often lower
 - iii) Females may be more selective (fewer partners, more selective).
 - (d) Medical changes
 - i) Metabolic
 - ii) Pain

IV. Conflict between dogs

A. Conflicting dynamics between animals.

- 1. Age/energy/size differences
 - a) Play/interaction need not met
 - b) Overly excitable
 - c) Trying to get needs met from the other animal
 - (1) Golden with senior pug
 - (2) Favoritism to puppy and making older dog tolerate
 - (3) Favoritism to older dog
 - d) Socialization issues
 - (1) Learning other breed's cues outside of select group
 - (2) Generalizing friend group to different sizes, breeds, play style, energy.
 - (a) Large dogs fearful of small dogs
 - (b) Aussie who stalked small dogs
 - e) Owner related
 - (1) Owner tension and reaction that causes escalation
 - (2) Competition over the person
 - (3) Person favoring one dog
 - (4) Punishment leads to anxiety and aggression
 - (a) Inhibits response doesn't change emotional state
 - (5) Dog not allowed to be a dog
 - (a) Small dog syndrome
 - (b) Too tolerant of a dog- Rockey dog
 - f) Dog not listening to the other's cues
 - (1) Asking for space, protecting space or item. Mutual motivation
 - (2) Dominance isn't a personality trait, it's a fluid interaction
 - (a) If dogs both properly motivated and neither one backing down, conflict may ensue.

B. Rivalry

1. Competition
 - a) How conflict is settled
 - b) Dominance
 - (1) Not a personality trait. Relevant within the interaction and fluid relationship.
 - (a) Dominance is rarely full of conflict
 - (2) Dominance (competition of first rights to a resource. Relationship interaction- not a personality trait. Most 'dominant' dogs are not that at all.
 - (3) One dog acquiesce, or equally motivated?
 - (4) Conflict over resources. Food. Toys. Chews. Resting spaces. Movement through hallways or door. Person. Attention.
 - c) Female rivalry. Sibling rivalry.
 - d) Same age/size/sex
 2. Changing roles
 - a) Aging dog.
 - b) Maturing pup.
 - (1) Less tolerance
 - (2) Entering sexual maturity
- C. Overexcitement/ overly aroused/ Redirection
 1. Overexcitement and redirected aggression (door, visitor, walks, meal, person home, leash, fence).
- D. Barrier frustration (leash/fence/car).
 1. Dog may learn they can't move away/approach.
 2. For some dogs more than just a display. Display only? Some dogs are aggressive on and off leash.
 3. What does the dog want? Space? Play? Root emotion often fear and frustration related.
 4. Signaling limited. Other dogs' body language changed by leash.
 5. May learn other associations (pain/punishment).
 6. Owner can escalate reaction (tight leash, anticipation, anger, picking dog up, avoiding altogether).
 7. Chained dog
 - a) Continued stressors
 - b) Other dogs can get there
 8. Invisible fence
 9. Negative experience.
 - a) Off leash dogs encountered.
- E. Other stressors
 1. Moving
 2. Home size
 3. Exercise/ mental enrichment
 4. New animals in the home
 5. Children
 6. New significant other
 7. Anxious over other situations
- F. Medical

1. Reproductive (females in heat, males).
 2. Possible medical and internal reasons
 3. Irritation from pain
 4. Medications
 5. Metabolic issues (thyroid)
 6. High protein in diet.
 - a) Chase instinct with dogs.
 - (1) Doesn't mean aggression (Gwen and dog).
 - (2) However, the cat doesn't understand.
 - (3) Cats resource guarding
 - b) Dog/Cat
 - (1) Chase, multiple animals?
 - (2) Predatory (stop on predatory sequence?)
- V. Conflict with dogs and cats
- A. Often about the 'chase'
 1. Most dogs not wanting to 'kill' the cat nor is the cat 'taunting and teasing' the dog
 2. If the cat stops, most dogs will stop with curiosity, interest and excitement. Not out to hurt.
 3. As excitement continues, it can build and may turn into a nip.
 4. Especially with people getting excited and seemingly chasing the cat with them.
 5. It can progress. Addressing early on is important.
 - B. Dogs and cats may not have been taught the other one is a 'friend' at an early age.
 - C. Introduction and how animals meet is important
 1. Early introduction may have gone awry or an experience caused major conflict (Asumi and cat running).
 - D. Preventing the chase is one of the most important aspects of keeping harmony.
 - E. Important to not let it continue even with dog who doesn't harm
 1. Risk still there
 2. Cat may become more fearful and withdrawn
 3. Dog can escalate in excitement and resolve to seek out those chases (stalk, watch)
 4. Cat may not make a stand
 5. Welfare (emotional/physical).
 6. Cat withdrawn from social situations
 7. Stress leads to other physical problems
 8. Cause conflict with people and their pets.
- VI. Risk factors and likelihood of a peaceable outcome.
- A. History
 1. Longer time more difficult to overcome
 2. Puppies and kittens more resilient and open to change
 3. Do animals have positive experience with other animals?
 - a) Especially important to look at adulthood experience.
 - B. Predictable triggers?
 1. Certain room or area of the home?

2. Certain people present?
3. Certain variables present?
 - a) Toys/chews/food bowl/treats/dropped food
 - b) Resting spaces
- C. Individuals involved
 1. The greater the number, the more difficult it may be to manage.
 - a) One or two pets may be the main problem
 - (1) Dogs fighting; Ryley and Shadow Lab were main combatants
 - b) With chasing, two dogs together may contribute to the problem.
 - (1) One chases and the other joins along.
 - c) Do animals get along with other animals?
 - (1) History of play, positive encounters?
 - d) Looking at each individual.
 - (1) Two cats were very different; one wanted to hide and was less sociable.
The other more social.
 - (2) Likelihood of success
 - (a) Gator was especially nervous with Labradors. Better with other breeds and played.
 - (b) Frenchie who aggressed at all dogs regardless of size, breed, personality.
 - (c) Dogs who react to younger males. Or, react to energetic, confidently forward dogs. Do better with laid back and more submissive dogs.
 - (d) Excitable dog who chased running cats, but lived well with calm cats.
 - (e) Dog constantly on alert and ready to chase small animals.
 - i) Dog on walks constantly scanning for bunnies, cats, joggers.
 - (1) High tendency to chase
 - (2) Practiced/rehearsed repeatedly
 - D. Longterm or short-term problem
 1. Temporary houseguest or living arrangement
 - a) Landlord with dog who fought
 - b) Deployment
 - c) Older dog
- E. Degree animals are affected
 1. Is it possible to get the animal's attention?
 - a) Take treats?
 - b) Play?
 - c) Interact with people? Enjoy normal activities?
 - d) Can you interrupt their focus? Redirect their focus?
 2. Are they obsessed overly focused on where the other animal is?
 - a) Way of life affected by the other animal
 - (1) Jumping at noises of other animal nearby
 - (2) Avoiding social situations.
 - (3) Hiding. Withdrawing.
 - (4) Confined to a certain area (okay?).

3. Recovery after an incident
 - a) Bounce back fast after upsetting incident?
 - b) How easy is it to keep animal's attention? Redirect focus?
 - c) How upset is the animal?
 - (1) Take treats with other animal around?
 - (2) Play?
 - (3) Getting enough sleep?
 - (4) Withdrawing?
 - (5) Signs of stress; lack of self care, hope accidents, jumpy/ on edge, preoccupation with where other animal is.
- F. Manageability
 1. On leash reactivity often much easier than in home dogs fighting.
 2. Home environment for separation/ training sessions.
 3. Time/resources of family
- G. Risk factors
 1. Warnings
 - a) Willingness to warn/move away rather than launch?
 - b) Warn multiple times?
 - (1) Body language multiple warnings vs. few
 - (2) Other dog's ability to understand and appropriately respond.
 - c) Predictable triggers?
 - (1) Calm, but if cat darts, then it's a problem.
 - d) Limited situations it occurs in?
 - e) Pattern worsening, staying the same, improving?
 2. Ability to interrupt
 3. Bites/ physical harm
 - a) Number of incidents
 - b) Bite inhibition
 - c) 'Snap' or 'muzzle punch'
 - d) Bite
 - (1) Depth.
 - (2) Number per incident.
 - (3) Grab and hold?
 - (4) Shake?
 - (5) Where bit?
 - (6) Number of animals involved
 - (7) Size difference.
 - (8) Another person's animal, or their own?
 4. Risks to people and dogs
 - a) Who is involved?
 - (1) Family? Other people's animals?
 - (2) Capability of people
 - (a) Physical ability
 - (b) Ability to manage situation
 - (c) What do they do in fights?
 - i) Able to react wisely?

- (1) Throwing themselves into the fight is seriously dangerous.
- (d) Ability to grasp concepts
 - i) Training
- (3) Other factors
 - (a) Physical capability to handle animals
 - (b) Shown wisdom with management and prevention?
 - (c) Children (age, personality)
 - (d) Roommates
 - (e) One partner more willing or able than the other.
 - (f) All people involved increases success. One may thwart efforts.
 - (g) Manageability of the home for prevention and separation.

VII. Goals/Expectations

- A. Expectations and reality of situation aligned?
- B. Too high for reality of situation?
 - 1. Addressing unrealistic expectations
 - a) Dogs to be best friends
 - b) Senior dog to have preference
 - c) Or, puppy to be 'played' with or not snarked at
 - d) May need other play friends
 - e) Dog to be friends with everyone
 - f) Cat and dog left unsupervised/ no guidance
 - g) Dog to be friends with everyone
 - h) Dog parks
 - i) Doggy daycare
 - j) Leash manners vs. off leash
- C. Expectations beyond what the family will contribute?
 - 1. Quick fix or committed longterm?
 - 2. Understanding management is a major piece of plan.
 - 3. Understand and able to live with risk?
 - 4. Invested fully in all pets involved?
 - 5. Resources (time, money, living arrangement) aligned with goal?
 - a) Time
 - (1) Work
 - (2) Other commitments (kids, pregnancy, moving, wedding, medical issues, care taking, other animals)
 - b) Financial resources
 - (1) Veterinary/Training help
 - (2) Tools
 - (a) Training tools
 - (b) Barriers (leashes, gates, crates)
 - (c) Treats/toys
 - c) Resources of people
 - d) Living arrangement
 - (1) Yard?
 - (2) Multiple rooms?
 - (3) Construct management and living space as needed?

D. Separation

1. Temporary separation protocol
 - a) Safety
 - b) Prevent rehearsal of unwanted behavior
 - c) Time for both animals to destress and relax
 - (1) Gradual reintroduction
 - d) Separation and management for shortterm situation

E. Longterm separation possibility

1. Separation when not supervised
2. Needing supervision together

F. What if's

1. If it doesn't work, are there ways to manage it?
 - a) Is separation okay? Does it offer a quality of life? Risk minimized?
 - b) Cat confined by choice or necessity. Is cat okay with it? Getting needs met?
2. Rehoming

VIII. Management and safety protocols

A. Safety measures and management to prevent rehearsal of unwanted behavior and lower risk

B. Safety equipment:

1. Muzzle (V)
2. Gates
3. Crates
4. Getaway spaces (rooms).
5. Breaking up altercation (noise, compressed air, citronella spray, bite stick, umbrella, push and pull by backlegs, two people, air horn, rape alarm, harness/head halter with dragline, doorbell/door/car distraction- ultimate goal for training to be way to breakup/redirect).
6. Avoiding conflicts unless training
7. Safe separation protocols:
 - a) Drag lines
 - b) Visual blockers/Distance
 - c) On leash (no retractable).
 - d) Waist leash
 - e) Tether while supervising during exercises (gate)
 - f) Harness (front clip)
 - g) Head halter
 - h) Come with me kitty harness
8. Getaway spaces
 - a) Cat room.
 - b) Escape/dwelling areas: Furniture (couch, bed), high spaces
 - c) Able to reach necessities (litter, food, sleeping areas, social time with family).

C. Preventing rehearsal and problem situations

1. Door bell alternative
2. Mealtime separation
3. Picking up toys and chews
4. Teach on/off furniture- redirect to right bed.

5. Environmental and individual needs attune for with each animal.
 - a) Exercise
 - b) Predatory type play
 - c) Giving the dog a job (scent work, agility, food puzzle, train)
 - d) Play outlets to meet need of individual (chase, fetch, herding, tear apart/destuff toys).
 - e) Medication options (fear, anxiety, increase confidence and likelihood to stay vs. flee, predatory behavior)

IX. Importance of signaling and monitoring interactions

- A. Learning to recognize early signs.
- B. Training humans to catch body language early (like train leaving tracks, longer it goes gains momentum and harder to stop). Early warnings vs. progressed.
- C. Not punishing the warning or not heeding a dog's discomfort and letting the dog endure the situation.
- D. Those who've learned signaling doesn't work or they've been punished for signaling, reinstalling (sniffs, moving away, snap, growls, lip lifts).
- E. Ample warnings? Have certain throngs in ladder been taken out? Or, has dog learned nothing else works to get their point through?
- F. Recognizing early signs before it progresses.
- G. Signs of discomfort/Fear/Anxiety
- H. Warnings and progression of aggression.
- I. Defensive aggression (fear)
- J. Offensive aggression (frustration/ agitation/ root may be fear but learned bigger displays work)
- K. Over arousal/excitement
 1. Watch the tail and ears up and rigid. Tight tail wag.
 2. Elevated movement and posture.
 3. Tense muscles.
 4. Vocalizations (barking, yips)
 5. Momentary pause
- L. Predatory/ Targeting as object of chase
 1. Staring/ Intense focus
 2. Stalking
 3. Stiff
 4. Raised tail
 5. Crouched
 6. Rush
 7. Down or standing position (sight/stalk)
 8. Multiple dogs with another dog or cat
 9. Fast, not slowed and bouncy. Efficient movement.
 10. Ears forward and rigid. Tail up and high or out and stiff.
 11. Quiet
- M. Play behavior
 1. Paw slap
 2. Dog laugh
 3. Hump

4. T-stance myth
5. Play bow
6. Self handicapping
7. Bouncy
8. Mixed signals
9. Changing roles or agreement on roles kept
10. Intent by both individuals to play
11. Breaks
12. Signaling intent of play may fade a little once dog is comfortable after the introduction (lessened play bow or shoulder roll).

N. Problems in play

1. Expectation good dogs play and happy dogs get to play.
2. Lacking realization of a problem (dog plays fetch, but doesn't interact with other dogs. Or, dog runs and comes to owner, but not actually soliciting play. Or, pug that goes to park and doesn't react, but constantly shaking off the stress after dogs interact with him).
3. Size difference problems (unintended injury, predatory drift).
4. Escalation of interest or excitement putting body in a state of heightened alert and stress (even good stress can put body in state where they're ready to react).
5. Not enough breaks to calm down (young kids playing when tired and someone gets hurt).
6. Dogs not always socialized with other breeds/size/play style. Rush attack. Paws and body slams. Herding.
7. Uneven matches in play
8. Targeting a certain dog
9. Dog cornered
10. Too fast, heightened, rough, not enough of a break, long chase sequence.
11. Size difference
12. Acting like prey (fluffy white dog with sighthound or with border collie mix).
13. Problems at the dog park: Unsocialized dogs brought to 'socialize'
14. Dogs not exercised prior
15. People not watching.
16. Dog used to playing roughly and not able to adjust play style and force.
17. Owner correcting with punishment creates a potential problem (shock collar/choke chain/ alpha roll).
18. Dog has select play partners rather than friends with everyone.
19. Dogs not heeding warnings to back off or lack of play signals.
20. The dog signaling not having their communication listened to.
21. Overly excited greetings or frustration with barrier at gate.
22. Dogs rushing the gate

X. Behavior Modification

A. Gradual exposure

1. Exposure at a distance
2. Visual blockers
3. Movement vs. still
4. Color/size/energy level/gender

5. Location (territorial behavior more likely at home or own neighborhood vs. unfamiliar place.)
6. This way
7. Low traffic times
8. Practicing with dogs less likely to cause a reaction
9. Distraction- find it, toy- stick.
10. Another outlet (run with person, toy fetch, stick toy for poodle mix).
11. Redirecting focus
 - a) Find it
 - b) Toy
 - c) Chase
 - d) Stick toy for poodle mix on walks when reacted at other dogs.
 - e) Fetch with Lloyd
 - f) Behavior asked to do (heel, sit, watch)
 - g) Teaching dogs to take breaks in play before escalation or if potential problem.
 - h) Avoiding leash introductions for the most part. If using, have a release cue to say hi. Leashes loose and circling with them to prevent tangling.
 - i) Timeouts for dogs being too exuberant (drag line potential)
12. Improving emotional reaction around the other animal
 - a) Teaching a new association: cc and neurochemicals.
 - b) New patterns and pathways in the brain with association.
 - c) Emotional reaction goal joyful anticipation
 - (1) Good things happen when the other animal Treats
 - (2) Toys
 - (3) Check ins. Start with sight of thing before animal reacts.
 - (4) Hear that. Tags jingle. Dog bark.
 - (5) One dog gets good things, that signals good things for me too (like resource guarding, pair a positive with other dog coming near).
13. Teaching new way to communicate
 - a) Being your dog's advocate important (remove before they escalate, don't punish the growl)
 - b) Reward of distance or getting to greet
 - c) Giving dog control over outcome.
 - d) Signals to meet end goal for the dog (space, distance, end of interaction, or for some, play and ability to interact).
 - e) Examples of some signs of conflict avoidance to reward for:
 - (1) Head turn
 - (2) Lip lick
 - (3) Shake off
 - (4) Sniff
 - (5) Turn away body
 - (6) Sit/lay down
 - (7) Mouth closed to open (about to bark or growl to letting out stress, open smile or pant).
 - (8) Watch the animal and see what they do that can be rewarded for (Bailey at kennel).

- (9) Fearful animals (cat or dog) rewarding confidence and calm behavior:
Ears forward from back position, tail raising, sit, down.
- 14. Teaching new habitual response
 - a) Replace other behavior before it takes place or asked for when redirected.
(refine to 3)
 - (1) Leave it
 - (2) Hand target
 - (3) Recall (during interaction, before released).
 - (4) Mat training/ Go to spot
 - (5) Sit/Down
 - (6) Eye contact
 - (7) Heel
 - (8) Check ins (seeing the thing and looking at person)
 - (9) Turn behavior and moving away from threat
 - (10) Drop it
 - (11) Say hi/ go play cues for those needing play.
 - (12) Interruption/ preventing rehearsal (front clip harness/head halter with pressure to sit, clap or stomp, abandonment or non reaction technique from their person).

XI. Successful outcomes

- A. Dakota Shiba puppy.
- B. Bully breed puppy in puppy class
- C. Over exuberant lab pup in class
- D. Riley
- E. Management of landlord dog
- F. Jack

Forelimb lameness: Diagnostic modalities

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Osteochondritis dissecans (OCD) is a manifestation of osteochondrosis in which a flap of cartilage is lifted from the articular surface. Osteochondrosis is thought to precede OCD and is a disturbance in endochondral ossification. The disturbance in endochondral ossification leads to multiple areas of cartilage islands on the surface and within the humeral epiphysis which have not undergone normal maturation into bone. Areas of abnormal endochondral ossification of the articular surface become thickened and are susceptible to fissure and loosening (OCD) as the deeper chondrocytes undergo necrosis due to inadequate nutrition and a suboptimal microenvironment.

Large and giant-breed dogs are commonly affected and males are more often affected than females. Clinical signs often develop between 4 and 8 months of age; however, some dogs may not be presented for veterinary evaluation until they are mature. Affected animals are usually presented for examination because of unilateral forelimb lameness. Owners usually report a gradual onset of lameness that improves after rest and worsens after exercise.

On physical examination, the shoulder should be palpated and moved through a complete range of motion. Crepitation or palpable swelling of the joint is seldom evident, but affected animals usually exhibit pain when the shoulder is moved into hyper-extension or extreme flexion. Often the examiner can detect muscle atrophy of the forelimb by loss of muscle mass adjacent to the spine of the scapula.

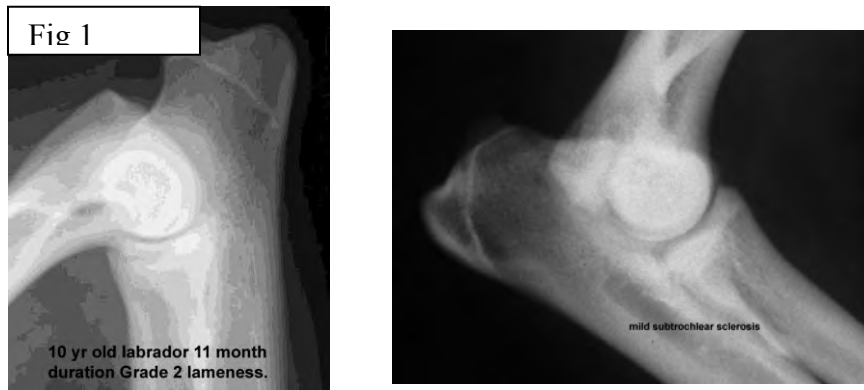
Radiographs: Despite apparent lameness in only one limb, both shoulders should be radiographed because this condition is often bilateral. Sedation may be required for quality radiographs particularly in large hyper-active dogs. The earliest radiographic sign of OCD is flattening of the caudal humeral head. This is due to thickening of the articular cartilage and deviation of the subchondral bone line.

As the disease progresses, a saucer-shaped radiolucent area in the caudal humeral head may be visualized. Calcification of the flap may allow visualization of the flap either in situ or within the joint if it has detached from the underlying bone. In chronic cases, large calcified joint mice are often observed in the caudoventral joint pouch or cranially within the bicipital groove.

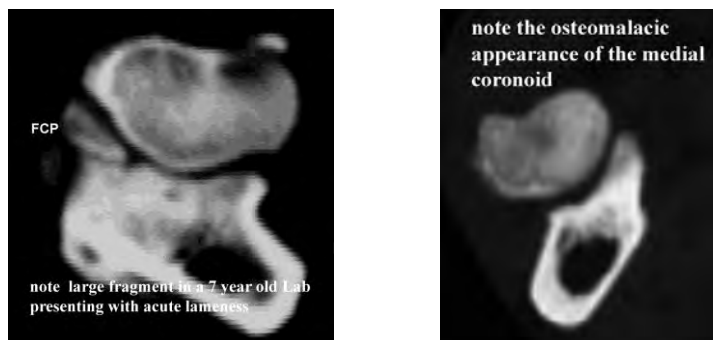


Forelimb lameness can be a diagnostic challenge in the athletic dog; often the lameness has been treated for months with no improvement. The only abnormal physical finding may be the observation of Grade 2 or Grade 3 lameness. The source of lameness may be

attributed to soft tissue injury, bony injury or a combination of both. In the active adult dog the most common cause of latent forelimb lameness can be attributed to pathology in the elbow and to injury of the active and passive shoulder restraints. In the author's experience, pathology in the elbow is regularly caused by occult microfracture/fragmentation of the medial coronoid process. There is no joint effusion, loss of motion, pain, or crepitus on physical examination. Radiographs are reported as normal or may show minimal subtrochlear sclerosis of the ulna.

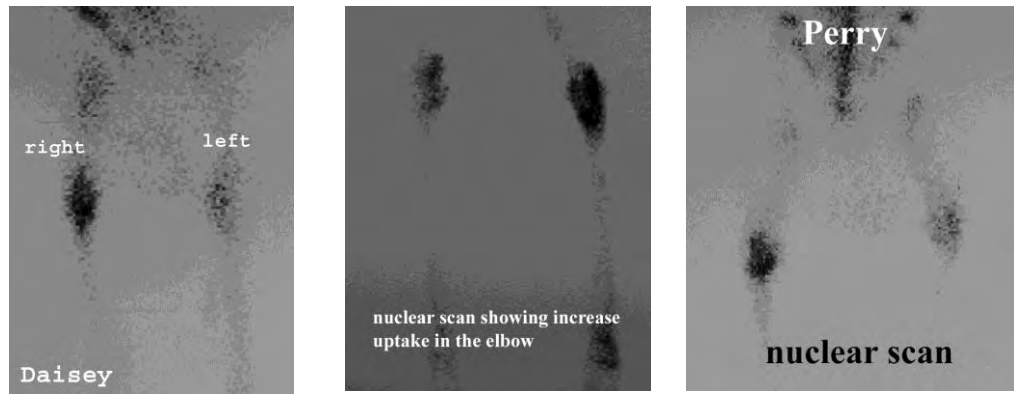


Modalities to facilitate an accurate diagnosis in these cases are CT, nuclear scan, and arthroscopy. Recommendations for performing CT include scanning from the point of the olecranon to 2cm distal to the radial head. Scan thickness should be 1-2mm with .5mm overlapping slice index. Transverse slices using 1500 to 3500 HU are ideal for imaging subchondral bone and fragments of the medial coronoid; transverse images at 3500HU are considered ideal for identifying osteomalacic lesions of the medial coronoid.

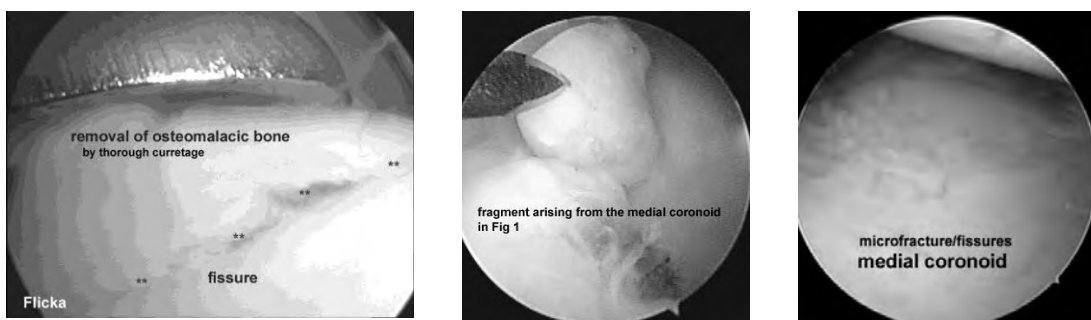


Nuclear scintigraphy can be used to localize the origin of the lameness and can be used to facilitate detection of subtle pathologic changes before changes are evident of radiographs. Technetium phosphonates are typically used for scintigraphy of joint tissues. Scintigraphy has high sensitivity for detection of presence of elbow pathology but is not specific for definitive diagnosis. Regulatory issues often limit the use of scintigraphy to academic institutions or large referral practices. Nevertheless it is invaluable facilitating lesion localization in dogs with forelimb lameness. Most commonly, it is used to rule in

or rule out elbow pathology. Note in the cases shown below the uptake in technetium in the involved elbow compared to the normal elbow. Each of the below cases had a long standing undiagnosed forelimb lameness. Radiographs of these cases were considered within normal limits. The use of scintigraphy localized the lesion to the elbow which then allowed application of more specific diagnostic modalities such as CT or arthroscopy.



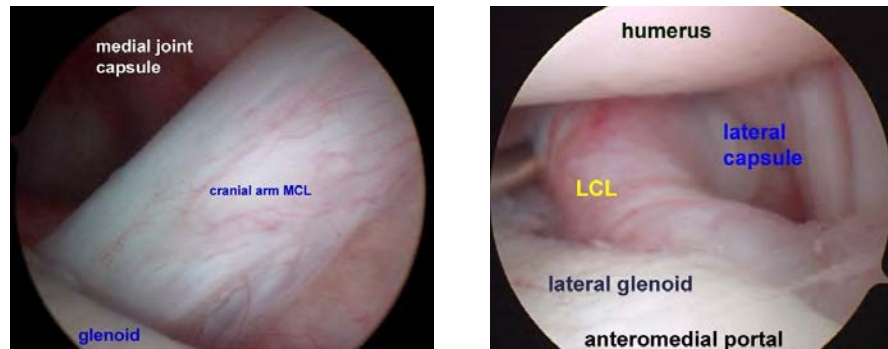
Arthroscopy is more invasive than imaging modalities but is very specific for identification of pathology in the medial compartment of the elbow. A comparison of CT with arthroscopy showed that these procedures were complimentary for medial coronoid assessment. Care must be exercised when assessing the medial coronoid on CT and arthroscopically. Fragmentation of the articular cartilage, micro fissures and nondisplaced fragments may not be detected on CT. Likewise, with arthroscopy, thorough probing and or curettage adjacent to the radial head often will reveal abnormal bone or fragments beneath the cartilage surface not visualized on with casual observation.



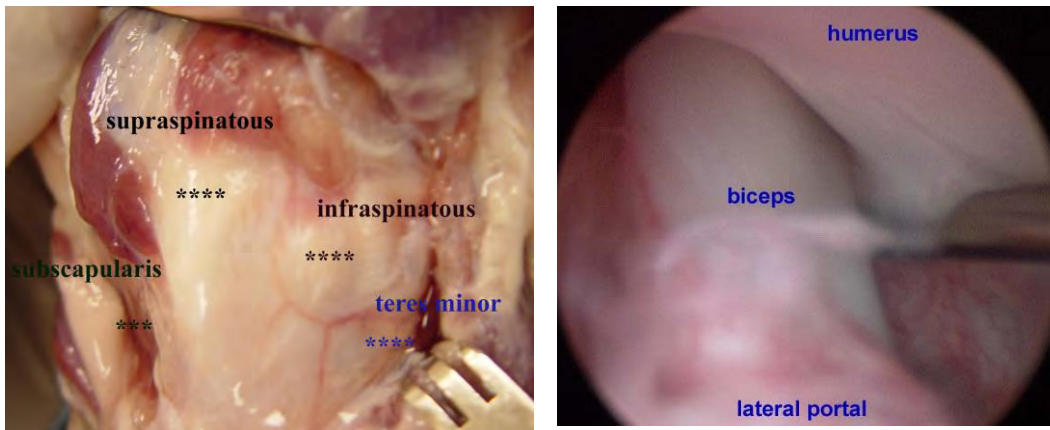
Active and passive restraints of the Shoulder: Diagnosis and Treatment

Forelimb lameness attributed to shoulder instability is reported frequently in the literature. Some orthopedic centers report the presence of subtle shoulder instability frequently where as others rarely diagnose the condition. The widespread difference in

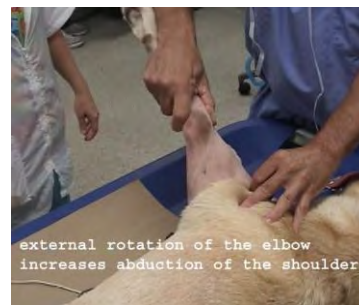
the frequency of diagnosis between clinics has led to disagreement relative to criteria for diagnosis of shoulder instability and the arthroscopic appearance normal intra-articular structures versus pathologic lesions. Stability of the shoulder is derived from passive and active restraints. Passive mechanisms include the medial and lateral glenohumeral ligaments, surrounding joint capsule, joint conformation, and synovial fluid cohesion. The medial collateral ligament (MCL) commonly appears as “y” shaped with the cranial arm coursing caudally from its origin at the medial surface of the supraglenoid tubercle. The caudal arm of the MCL originates from the medial surface of the scapular neck and joins the cranial arm to insert onto the humeral neck. The MCL and associated joint capsule is a major factor in providing joint stability; complete medial luxation occurs following transection of the MGHL. The lateral collateral ligament (LCL) originates from the lateral rim of the glenoid and extends ventrally to insert onto the humerus at the caudal region of the greater tubercle. The joint capsule originates from the periphery of the glenoid cavity. Medially, the joint capsule forms a synovial recess due to its attachment several millimeters proximal to the glenoid rim.



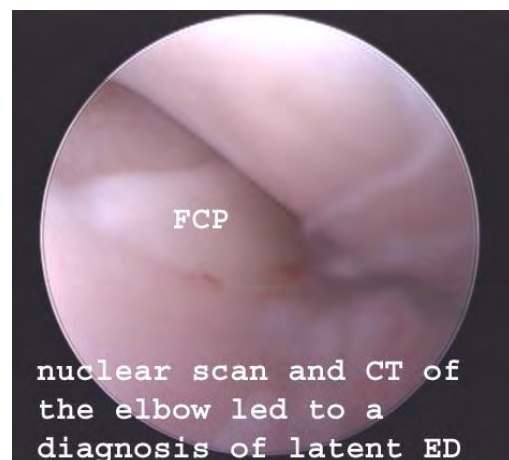
Dynamic active glenohumeral stability is provided by contraction of the surrounding cuff muscles. These include the biceps brachii, subscapularis, teres minor, supraspinatus, and infraspinatus muscles. Active contraction of all or selective cuff muscles induce compression across the shoulder joint as well as increasing tension in the joint capsule. When tested in neutral position, the cranial, lateral, and medial translation of the humerus was significantly increased after biceps tendon transection. In the flexed position, translation of the humerus in the cranial and lateral directions was significantly increased after biceps tendon transection. In the extended position, the medial translation of the humerus was significantly increased after biceps tendon transection.



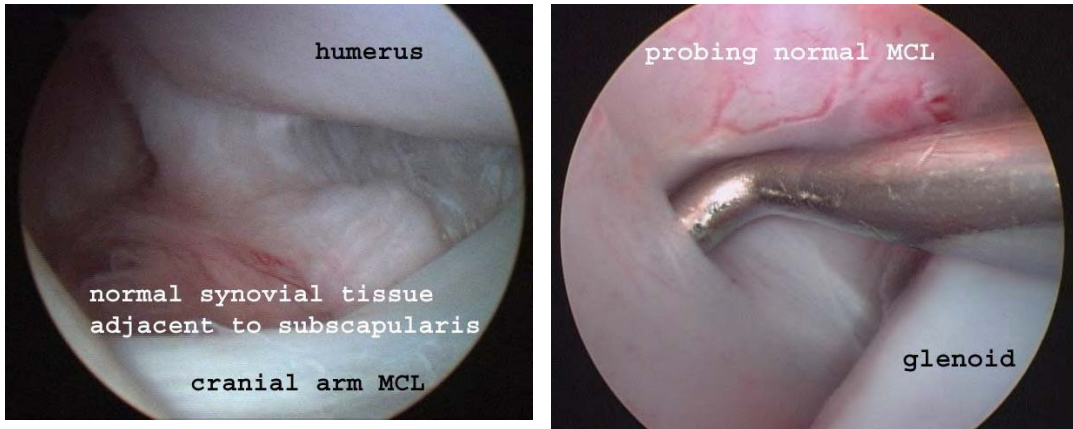
Examination of the shoulder for stability should be done under anesthesia or heavy sedation. Flexion, extension, abduction, craniocaudal translation, and rotational stability of the shoulder joint should be assessed. Normal range of flexion and extension are 40 degrees for flexion and 165 degrees for extension. Circumduction of the shoulder should not give rise to subluxation. Anteroposterior translation should be similar in both shoulders. A normal abduction test is approximately 23 degrees; abnormal abduction is considered present when abduction exceeds this degree and there is a difference in abduction angle between the injured side and the normal side. When performing the abduction test, it is essential to maintain the limb in extension with the elbow in neutral position, ie, one does not want the elbow externally rotated. If the elbow is externally rotated with the limb in extension, the shoulder joint will be internally rotated. The latter will give a false positive abduction test. To maintain the elbow in neutral position, the examiner should place his/her thumb on the lateral surface of the olecranon just posterior to the humeral epicondyle. Maintaining the thumb facing upward assures that the elbow remains in neutral position.



Care should be exercised in interpretation of side to side laxity difference. We have documented consistent abnormal abduction and AP translation in cases of long standing unilateral forelimb lameness. Our interpretation of shoulder laxity in these cases is that muscle and ligamentous atrophy give rise to increased shoulder laxity. In some cases, the shoulder laxity is secondary to long standing shoulder pathology such as OCD. Other cases are referred to our centers for treatment of shoulder pain. The pain is noted when the shoulder is placed in extension. By and large these cases are latent elbow dysplasia; the pain in fact arises from extension of the elbow when the shoulder is placed in extension.

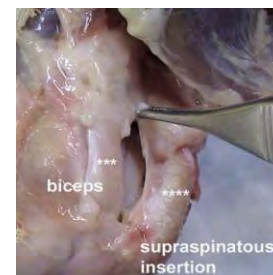
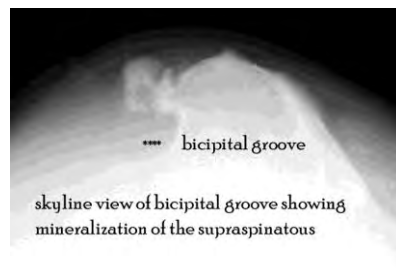


Debate continues amongst surgeons as to the interpretation of the arthroscopic appearance of intra-articular structures. Some surgeons believe it is possible to detect increased intra-articular volume and redundant capsular/ligament structures within the joint while others believe this cannot be accurately detected arthroscopically. Further, the significance of intra-articular lesions remains controversial. While some surgeons believe small tears of the cranial arm of the MCL result in instability and require treatment, others believe these small tears are insignificant and most likely iatrogenic. The gutter medial to the cranial arm of the MCL adjacent to the insertion of the subscapularis is often diagnosed as a site of trauma. However, this zone often shows synovial proliferation with pathologic conditions of the shoulder such as OCD.



Treatment of Forelimb Disorders
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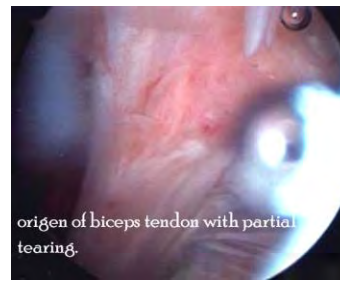
Supraspinatus strain: Dogs with strain of the supraspinatus tendon present with a chronic foreleg lameness; some dogs will exhibit periods of non-weight bearing lameness. Although uncommon, any age or breed of dog can be afflicted; however, the condition is seen more in large breeds of dogs (Labrador, Rottweiler). Radiographic views should include standard lateral projections of both shoulders. Mineralization is seen adjacent to the greater tubercle of the humerus. Patterns of mineralization are either irregular, non-homogeneous or well circumscribed and dense foci. A “skyline” view of the bicipital groove is helpful to delineate the location of dystrophic mineralization.



It is worthy to note that the mineralization of the supraspinatus insertion indicating chronic strain may be present but not be the cause of clinical dysfunction. Often mineralization is present in the shoulder in which the dog is not lame. Bilateral mineralization may be present (although different stages of mineralization can be seen between shoulders) and the dog only lame in one shoulder. Recently, MRI has been advocated as a diagnostic tool for shoulder lameness. Some surgeons believe that mineralization of the supraspinatus tendon displaces the biceps tendon causing biceps tendon pain. Although possible, ultrasound examination of the biceps tendon does not demonstrate inflammation secondary to biceps impingement. Also, the position of the biceps tendon is 3-5mm separate from the insertion of the supraspinatus tendon on the greater tubercle of the humerus. Arthrography can be used to outline the bicipital groove to determine if irregularities or filling defects suggestive of bicipital tenosynovitis are present. Diagnosis is based on clinical signs, imaging, and most importantly, ruling out other causes of forelimb lameness. Treatment is enbloc resection of the chronically inflamed section of the tendon. The tendon can be tenodesed in a position where it is exposed to less strain.

Bicipital tenosynovitis: Bicipital tenosynovitis is an inflammation of the biceps brachii tendon and its surrounding synovial sheath. The etiology of bicipital tenosynovitis is either direct or indirect trauma to the bicipital tendon or tendon sheath. Direct trauma due to repetitive injury may be an inciting factor and result in partial or complete tearing of the tendon. Indirect trauma secondary to proliferative fibrous connective tissue, osteophytes or adhesions between the tendon and sheath limit motion and cause pain. It has been hypothesized that mineralization of the supraspinatus tendon causes a secondary

mechanical bicipital tenosynovitis. Affected dogs are usually medium- to large-sized, and middle-aged or older. Working and active dogs are more commonly affected; there is no predisposition for either sex. Intermittent or progressive forelimb lameness, which worsens after exercise, is common. The owner may relate the lameness to trauma, but usually there is slow onset of clinical signs. Radiographically, bony resorption at the supraglenoid tuberosity is characteristic of chronic strain at the origin of the biceps tendon. Medical or surgical management have been successful in treatment of bicipital tenosynovitis.

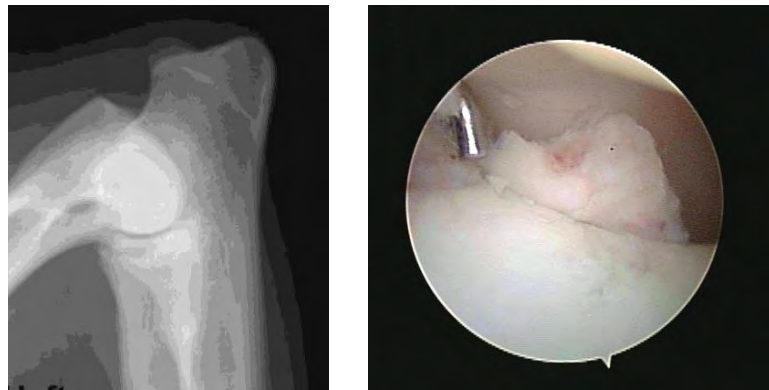


Medical treatment consists of injecting methylprednisolone acetate into the tendon sheath and restricting activity for 3 weeks. In one report (Stobie, JAVMA 1995) reported 50% of dogs treated medically had good to excellent outcome. In the same report, all dogs treated surgically had good to excellent outcome. Given these results, initiating treatment with 1 to 2 steroid injections is a reasonable approach. If outcome is not favorable, surgical intervention is advised. The bicipital tendon may show partial tearing of tendon fibers or partial tearing of the origin of the biceps tendon at the supraglenoid tuberosity. If the biceps tendon shows evidence of partial or complete tearing, the tendon is released by transecting the tendon just distal to the torn portion. Likewise, if there is evidence of synovial proliferation, mineralization, and osteophyte formation within the bicipital groove, the tendon is released from its origin. Tendon release may be performed with either a blade instrument (banana knife, beaver blade, or 11 blade) or a radiofrequency probe. The blade is slightly faster than the radiofrequency probe; however, use of the probe will prevent problems with hemorrhage from vasculature that is often present in the center of the tendon. Upon completion of tendon release, the tendon origin should be closely inspected for small osteochondral avulsion fragments, which can be removed with a motorized shaver or graspers. Lastly, the joint is thoroughly flushed by increasing the ingress flow and allowing egress through a large instrument cannula. Inspect the joint for remaining pathology and then remove the arthroscope and instrument cannula. Suture the portals with non-reactive, non-absorbable suture.

Elbow: Medial compartment syndrome has been classically attributed to elbow dysplasia. The term canine elbow dysplasia is used to denote an abnormal development of the elbow joint which results in a degree of incongruence. It is used to describe developmental diseases which include ununited anconeal process, fragmentation of the medial coronoid process and osteoarthritis, all believed to be secondary to joint incongruity. The proposed incongruence results in mechanical overload of the medial coronoid/medial humeral condyle through establishment of a “short” radius or “long” ulna. The fact that

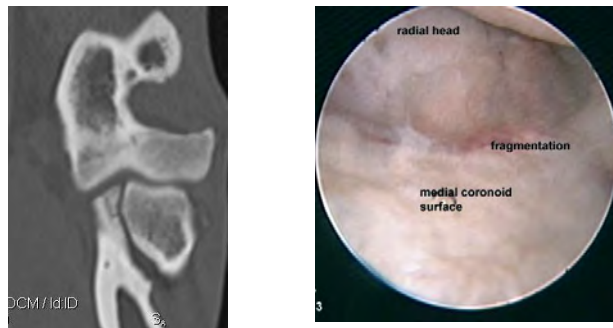


fragmentation of the medial coronoid will occur with incongruence is well noted in cases of concentric distal radial physeal injury. The latter results in a short radius, mechanical overload of the medial coronoid, and microfracture/fragmentation of the medial coronoid. Does elbow dysplasia (incongruence) exist? Yes. No question elbow dysplasia is seen in younger dogs with developmental incongruence. Not the severe cartilage change and fragmentation of the medial coronoid in the young Irish Wolfhound in the figure. However, elbow incongruence is not present in all dogs with elbow dysplasia; perhaps we do not have the imaging modality to detect subtle incongruence. Also, elbow dysplasia, a developmental condition that should be present from a juvenile age, does not account for the senior dog who has never experienced a lameness problem and presents with sudden onset lameness. The latter signalment/presentation accounts for the majority of forelimb lameness cases attributed to the elbow encountered by the author. As such, there must be other factors involved in the development of medial compartment syndrome.



9 yr old Malinois with acute onset lameness: note fragmentation, no articular cartilage pathology

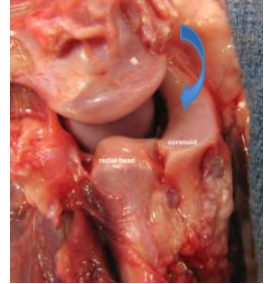
Fitzpatrick (proc ACVS 2010) has shown with the use of micro CT that the pattern of trabecular fracture is consistent with acute traumatic overload (cranial tip fragment) or chronic rotational overload (radial incisures fracture). Recent work by Goodrich (proc VOS 2013) clearly has shown the weight bearing axis of the forelimb to be directed through the medial compartment. Also evident through CT of cases with medial compartment syndrome there increased bone density (sclerosis) in the medial compartment consistent with chronic repetitive mechanical overload. Acute mechanical overload directed through the medial compartment could easily account for the cranial tip fractures as described by Fitzpatrick.



9 year old Retriever with sudden onset lameness; collapse medial compartment

Repetitive mechanical overload generated by years of athletic or playful activity can account for the fragmentation and wear of articular cartilage.

Fitzpatrick has also proposed repetitive rotational overload as a possible cause of radial incisure fragmentation of the medial coronoid. The exact cause of the rotational load is not known but may be attributed to muscular forces (ex. contraction of the biceps/brachialis complex, Hulse Vet Surg 2010) or rotation of the medial humeral condyle against the radial incisure of the medial coronoid (Bottcher proc ACVS 2013). repetitive rotation of the medial coronoid by contraction of the biceps/brachialis complex compresses the coronoid against the radial head generating a shear stress Which corresponds to the fracture plane of a radial incisures fracture



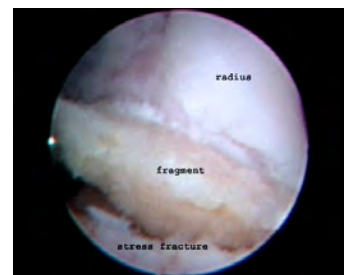
Peter Bottcher has described rotation of the medial humeral condyle On the radial incisures as a possible cause of fragmentation.



Treatment

Treatment of medial compartment syndrome includes fragment removal. This includes not only visible fragmentation but fragmentation which may be present beneath the articular surface (subtotal coronoidectomy). If surface articular pathology is present (abrasion) further treatment with medial compartment arthroplasty (abrasion arthroplasty, microfracture), decreasing medial compartment mechanical load (biceps release, sliding humeral osteotomy), and conservative modalities (weight loss, O- 3 FA diet, exercise moderation, nutraceuticals, ACP, stem cell, adequan, polyglycan, NSAIDs) are indicated. If clinical lameness persists, joint replacement (CUE, Tate TER) are considered.

Fragment removal is achieved via arthroscopy or mini-arthrotomy. It is important to note that stress fractures may be present in addition to the obvious visible fragment. A limited subtotal coronoidectomy is advised. A radial incisure fragment in an adult Pittbull with sudden onset lameness. 1 yr prior to this surgery, a small fragment was removed. Dog never recovered. Repeat arthroscopy found this fragment beneath the articular surface.



Mechanical unloading of the medial compartment:

Biceps ulnar release procedure;

The biceps/brachialis muscles constitute a large muscular complex. The anatomic origin and insertion of the biceps and brachialis muscles are such that the muscular complex exerts considerable force on the medial compartment of the elbow. The force exerted by the biceps is continuous since it is a pennate muscle with central tendon. More importantly, because the insertion of the biceps/brachialis complex is at the ulnar tuberosity, a large polar (rotational) moment is exerted at the cranial segment of the medial coronoid. The magnitude of the polar moment is a product of the moment arm (distance from the ulnar tuberosity to the tip of the coronoid) multiplied by the force created by the biceps/brachialis muscular complex. The polar moment rotates and compresses the craniolateral segment of the medial coronoid against the radial head. The compressive force is medial to lateral transverse to the long axis of the coronoid. A compressive force generates internal shear stress at an oblique angle to the applied compressive force. In this situation, maximal internal shear stress would be oblique to the long axis of the coronoid. Under the right circumstances, the polar moment and resultant compressive force produced by the biceps/brachialis complex may produce sufficient internal shear stress to exceed the material strength of the cancellous bone in the craniolateral segment of the medial coronoid. The result would be microfracture/fragmentation adjacent to the radial head at an oblique angle to the long axis of the medial coronoid. The surgical technique involves releasing the ulnar insertion of the biceps to unload the medial compartment and prevent the rotational moment rotating the coronoid into the radial head.

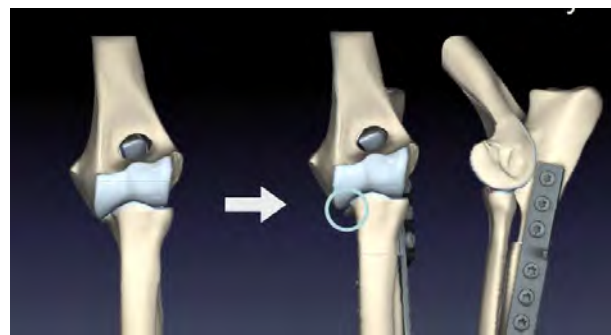


Osteotomies: The deviation of the mechanical axis medially and the humeral varus become more apparent with the progression of medial compartment OA similar to the process in the human knee. The result of overload of the medial compartment, collapse to the medial compartment, and OA. Sliding humeral Osteotomy(SHO): Sliding humeral osteotomy involves creating a midshaft transverse humeral osteotomy and translating (sliding) the diaphysis distal to the osteotomy medially. Doing so shifts the weight bearing axis through the elbow joint from the medial compartment to the lateral compartment. Owner and vet VAS scores have improved in all cases with a notable decrease in pain upon elbow manipulation

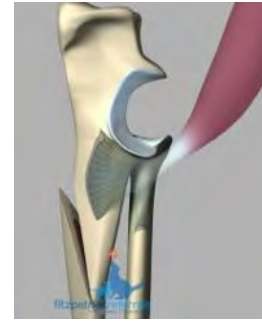


PAUL procedure

The PAUL procedure shifts the weight bearing axis through caudal tipping of the medial coronoid

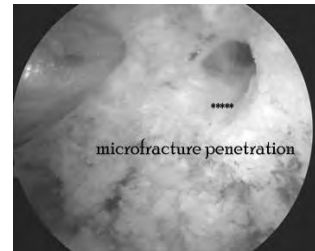
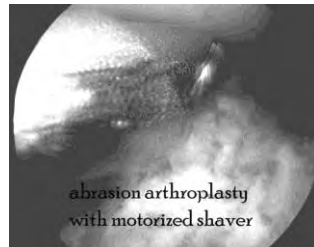


Dynamic bi-planar ulnar osteotomy described by Noel Fitzpatrick as a method of unloading the medial compartment.



Arthroplasty

Management of articular cartilage lesions is based on the concept that providing blood with mesenchymal cell precursors access to the lesion; this encourages healing by formation of fibrocartilage. Several marrow stimulating techniques have been described to achieve this. Abrasion arthroplasty involves uniform removal of subchondral bone until bleeding is achieved. This can be accomplished in the canine elbow by use of either a curette or burr attachment on a small joint shaver. The shaver is usually more rapid and efficient and generally just as accurate. Another marrow stimulating technique is microfracture. In this technique numerous microcracks are created in the subchondral bone plate with a specialized micropick to allow bleeding at the lesion surface.



Objective evidence documenting the efficiency of abrasion arthroplasty or microfracture is not available in the dog. The figure to the right shows resurfacing of the medial coronoid in a dog 6 months after abrasion arthroplasty. In man microfracture appears to be more effective than abrasion arthroplasty and is the marrow stimulating technique of choice. The technique is highly dependent on appropriate post operative rehabilitation. In man, 4-6 weeks of non-weight bearing activity coupled with active or passive range of motion is necessary for ideal outcome. Overall, the results of abrasion arthroplasty have been unpredictable and symptoms often recur 2-3 years after surgery. Nevertheless, good to excellent results are reported in 50 – 60 % of patients.



Elbow replacement is an option in dogs which have end stage elbow OA and conservative/less invasive surgical modalities have not resolved clinical pain. A number of prosthesis are available but the most popular one today is the TATE elbow. Clinical outcome studies indicate that a mechanical lameness may persist but that the dogs appear

to be less painful. A prosthesis presently in clinical trial is the CUE (canine unicompartamental elbow). The concept is simple and carries little morbidity. Information concerning this technique will be forthcoming in the near future.

TATE total elbow



CUE



Treatment Modalities for the Conservative Management of Osteoarthritis Don Hulse DVM, Dip ACVS, ECVS

Osteoarthritis is the most common cause of chronic pain in dogs with approximately one in five adult dogs having OA. OA (sometimes referred to as degenerative joint disease) is a slowly progressive degenerative disease involving the entire joint: articular cartilage, subchondral bone, synovial lining, joint fluid, ligaments, and muscles. Osteoarthritis is commonly classified as primary OA or secondary OA. Primary OA is associated with aging and chronic loading and wear of the articular surface. Secondary OA (the most common form seen in dogs) has many acquired and congenital etiologies including: ligamentous injury (CCL), abnormal joint conformation (elbow dysplasia), Osteochondrosis (OCD shoulder). In general, OA can develop in any joint where abnormal stresses are imposed on a normal joint or alternatively where normal stresses are imposed on an abnormal joint. Although more senior dogs exhibit clinical signs of OA as compared to their younger counterparts, younger dogs may also exhibit signs of OA. The most common example in younger dogs being OA associated with hip dysplasia. It becomes easy to understand why OA is painful when joint innervation and the role of inflammatory cytokines are considered.

Innervation of joints includes nociceptors which are free nerve endings found in all joint tissue except articular cartilage. They are found in the subsynovial layer only two to four cell layers beneath the synoviocytes lining the joint cavity. Dogs with OA have an ongoing synovitis the severity of which varies depending upon activity and joint trauma. The synovitis is accompanied by the accumulation of increased levels of eicosinoids (prostaglandins, leukotrienes) and pro-inflammatory cytokines (IL-1, TNF, NO) in joint fluid. Additionally, synovitis is accompanied by increased vascular flow in the subsynovial tissue. These two factors, increased inflammatory mediators in the joint fluid and increased blood flow in subsynovial tissue, increases the exposure of free nerve endings (nociceptors) to inflammatory mediators. The result is sensitization of free nerve endings, increased stimulation of free nerve endings, and transmission of pain to the CNS. Inflammatory mediators also up regulate the expression of harmful mediators which play a role in catabolism of articular cartilage.

The architect of cartilage is the chondrocyte which produces the extracellular matrix. The matrix is composed of glycosaminoglycans (hyaluronan and proteoglycan) and collagens (mainly type II). The collagen forms a dense network that retains the proteoglycan. The proteoglycan is highly charged and attracts water into the tissue. Thus cartilage is 75% water. In normal cartilage there is a very slow turnover of collagens but the proteoglycan is constantly being renewed. The proteoglycans are aggregated into large molecules ("aggrecan") with a protein core and many side chains of keratan sulphate and chondroitin sulphate. This core is in turn bound to hyaluronan chains with each chain containing many proteoglycan molecules. Aggrecan and water provide the compressive stiffness to the tissue whereas collagen provides the tensile strength. The morphological changes seen in OA include: 1. cartilage loss, especially in areas of increased load, 2. subchondral bone remodelling (loss of bone initially followed by sclerosis), 3. marginal osteophytosis, 4. variable synovial inflammation. The biochemical changes in the cartilage include: 1. loss of proteoglycan, 2. upregulation in the degradative and synthetic activities of chondrocytes, 3. disruption of the collagen network, 4. increase in water content. These changes reduce the elasticity of the cartilage leading to fibrillation and fissuring of the cartilage with eventual loss of tissue. If this continues eburnation of subchondral bone may result. It is proposed that the cytokines responsible for stimulating cartilage degradation in OA are

interleukins 1 and 6 (IL-1 and IL-6) and tumor necrosis factor- α (TNF- α). However, whilst these cytokines have been shown to stimulate degradation in several species, their effect in the dog is less marked. Recent in vitro studies (Innes) on canine cartilage explants show the resistance of canine cartilage to rhIL-1, rhIL-6 and rhTNF- α . However, canine cartilage does respond readily to oncostatin M (OSM) and Leukaemia Inhibitory Factor (LIF). Catabolic cytokines can stimulate the chondrocyte to produce and release degradative enzymes. The enzymes studied in most detail in this respect are the matrix metalloproteinases (MMPs) and the new family of endopeptidases the ADAM-TS-4 and -5 (**A** disintegrin and **metallo**proteinase with a **thrombospondin** motif). ADAM-TS-4 and -5 are also known as aggrecanases. MMPs and aggrecanases can cleave the protein core of aggrecan so as to release the majority of the molecule from the matrix. Under normal circumstances the chondrocyte also produces a natural inhibitor of these enzymes known as tissue inhibitor of metalloproteinase (TIMP). TIMP production appears to be decreased in OA.

Osteoarthritis progresses slowly and has a gradual onset of clinical signs. Subsequently, the diagnosis of OA is often made in the later stages of the degenerative process after extensive bone and joint damage has occurred. Commonly the diagnosis of OA is made by radiographic changes characteristic of degenerative joint disease. However, by the time radiographic changes are apparent the condition has progressed considerably. Therefore, early intervention using alternative diagnostic modalities is essential for the well being of the animal. One recommendation is to establish an Osteoarthritis pain assessment screening protocol. Behaviors consistent with OA in dogs include: limping, inactivity, difficulty rising, lagging behind on walks, stopping on walks, difficulty posturing to eliminate. Managing the osteoarthritic dog is multifocal; An accurate diagnosis is essential for the management of secondary osteoarthritis since surgical intervention may be necessary to correct the underlying problem to achieve optimal outcome. In addition to appropriate surgical intervention, successful treatment of osteoarthritis is a compilation of strategies including client education, behavior modification (both client and pet), appropriate exercise activities, rest, weight control, disease modifying agents and anti-inflammatory medications. Of these, controlled exercise activity coupled with adequate rest and weight control will benefit your pet as much or more than any other modality.

Regular physical activity and rest play a key role in wellness. Episodic physical activity may also be preferable to continuous exercise by avoiding injury due to overuse. Episodic activity refers to those activities that occur for a reasonable time period multiple times throughout the day. Of considerable harm to the process of osteoarthritis is your pet having a sedentary life throughout the week only to exercise strenuously on the weekend. This lifestyle exacerbates the osteoarthritis and is very likely to result in serious injury. Treatment regimes should include regularly scheduled rest. Exercise effectively squeezes the water out of the cartilage making it less compliant and more susceptible to injury. Rest allows fluids to seep back into the cartilage restoring its mechanical efficiency and lessening the incidence of injury due to overuse. Family members must learn to recognize their pet's body signals and know when to stop or slow down. Doing so prevents pain and injury caused by overexertion. Two types of exercise are important in osteoarthritis management. The first type, therapeutic exercises, keeps joints working as well as possible. Therapeutic exercises are low impact and designed to maintain or increase joint range of motion, proprioceptive feedback, muscle tendon unit and periarticular tissue elasticity. Examples of therapeutic exercises are passive range of motion activity, massage, aquatic therapy, and stretching. The other type of exercise, aerobic conditioning exercises, improves strength and fitness, and controls weight. Examples are brisk

walking, brisk, walking or trotting through high grass, cavaletti training, and aquatic therapy. Your veterinarian and/or a rehabilitation therapist can evaluate your pet and develop a safe, personalized exercise program to increase strength and flexibility. Each program will include a warm up period, exercise period, and cool down period. Weight and body condition are important in preventing Osteoarthritis as well as an important factor in the treatment of osteoarthritis. Heavy dogs are at increased risk of developing arthritis because their joints may be strained by excess weight. This is especially evident in weight-bearing joints such as the knees and hips, which often show the first signs of weight-related strain and injury. An investigation into the cause of cranial cruciate ligament injury and the development of secondary osteoarthritis showed a significant risk factor to be obesity. One study in man showed that an average of 10 pounds of weight loss over a 10-year period decreased the risk of osteoarthritis of the knee by 50%. Similarly, obesity accounts for up to 30% of knee OA in man, exacerbates symptoms, and is associated with more rapid progression of the disease. If your pet is overweight and you enforce a weight loss program, you will dramatically decrease the risk of your pet injuring its knee joint and developing osteoarthritis. In fact studies of dogs with hip osteoarthritis show that reaching target reduction weight increases a dogs' ability to move in a more normal fashion as assessed by gait analysis and owner observations.

Pain control medication allows the OA dog to engage in activity; this in turn helps control body weight and improve physical condition. The drugs of first choice for controlling arthritis are NSAIDs. NSAIDs function in part by inhibiting cyclooxygenase (COX) isoenzymes. COX-1 is the constitutive isoenzyme essential for the synthesis of homeostatic PGs in the GI tract, kidney, and platelets. COX-2 is for the most part induced and results in the production of PGs associated with pain and inflammation. However, COX-2 is also constitutive expressed and has a homeostatic role in canine brain, kidney, and vascular tissues. COX-3 is constitutively expressed and plays a role in brain tissue. NSAIDs approved for use in the dog include carprofen, deracoxib, etodolac, meloxicam, tepoxalin and others. All inhibit COX -1 and COX -2 to varying degrees. The Coxib-class may exhibit less interference with the homeostatic functions of PGs associated with COX-1. However, the clinical effect of COX 1 vs COX -2 inhibition is largely unknown (Vioxx!!)

Carprofen, a NSAID which is less ulcerogenic, is marketed by Pfizer Animal Health under their trade name Rimadyl™. Rimadyl relieves pain and clinical signs of osteoarthritis in dogs, while causing less gastrointestinal side effects. Plasma and serum concentrations of carprofen are consistent throughout the treatment period. Serum concentrations peak at 2 hours, while synovial concentrations peak between 3-6 hours. The synovial concentration of carprofen ranges between 1-10 µg/ml during the treatment period in both normal and osteoarthritic joints. A significant reduction of PGE₂ from chondrocytes occurs at all concentrations in this range. Recent studies have shown carprofen to have little effect on kidney and platelet function. Carprofen has been recently found to support cartilage metabolism and proteoglycan synthesis.

Etodolac (EtoGesic) is a Fort Dodge product used for treatment of osteoarthritis in dogs. The drug is available as a non-chewable tablet and is administered at a dose of 10-15 mg/kg every 24 hours. Etodolac has been found to be an effective treatment for ameliorating the clinical signs of osteoarthritis. Side effects with etodolac are typical of that seen with the NSAID class of drugs, gastrointestinal ulceration being the most common problem. Gastrointestinal ulceration can be severe at dosages above the labeled dose- this is well documented in their label claim during toxicity trials. Conflicting data has been found on

etodolac's effect on proteoglycan synthesis and cartilage metabolism. The Cox 2:Cox 1 ratio appears to be less favorable as compared to carprofen.

Meloxicam was granted USDA approval in 2003, having been available in Europe since 1993. It is indicated for the control of pain and inflammation associated with OA in dogs. It is considered to have moderate COX-2 inhibition.

Deracoxib (Deramaxx) is a recently released NSAID from Novartis Animal Health approved for use in dogs for postoperative pain and inflammation. The recommended dose is 3-4 mg/kg, po, once daily for 7 days or 1-2 mg/kg, po, sid for chronic use. Like carprofen, deracoxib has a highly favorable Cox 1:Cox 2 ratio. The expected side effects are similar to other NSAIDS, primarily gastrointestinal disturbances.

The first dual-pathway (cyclooxygenase, lipooxygenase) canine NSAID, tepoxalin, has recently been approved. It has been suggested that the reduced ulcerogenic activity of tepoxalin is due to the ability to inhibit leukotriene production.

For many years, Aspirin was the most common NSAID used in the dog. Although effective in the majority of cases, aspirin is COX-1 selective causing platelet dysfunction and GI toxicity. Nevertheless, empirical observation would suggest that as many as 40% of pet owners administer aspirin to their pets. Even low dose aspirin causes GI lesions in dogs. However, dogs develop a tolerance to aspirin and lesions do not necessarily worsen. This has recently been explained by production of endothelial cell triggered lipoxin. Aspirin triggered lipoxin (APL) appears to be anti-inflammatory and decreases PMN migration to areas of ulceration. The production of APL is mediated through the COX-2 pathway. If aspirin is followed by or given concurrently with a COX-2 inhibitor, the APL pathway is blocked. Rather than APL production, a different pathway occurs giving rise to leukotriene B4 which is a very potent inflammatory cytokine. The result is a significant increase in GI ulceration. The clinical message is that one should not administer aspirin with a COX-2 inhibitor or administer a COX-2 inhibitor without adequate washout if aspirin has been used (10 -14 days).

Chondroprotective agents are emerging as a new class of drugs used to slow progression of and treat chronic DJD. These drugs not only should be antiinflammatory; but also should support anabolic (repair) processes in cartilage, bone and synovium essential for normalization of joint function. This class of drugs include the glycosaminoglycans. Examples of these drugs include glycosaminoglycan polysulfate ester, pentosen polysulfate and sodium hyaluronate. Cosequin (Nutramax Laboratories, Baltimore, MD) is marketed as a glycosaminoglycan enhancer, capable of providing raw materials needed for the synthesis of extracellular matrix of cartilage. Unlike most nutraceuticals, Cosequin has been evaluated in a variety of studies. Cosequin contains glucosamine which has been described as the building-block of the matrix of articular cartilage. It has been described as a preferential substrate and stimulant of proteoglycan biosynthesis, including hyaluronic acid and chondroitin sulfate. Cosequin also contains chondroitin sulfate, mixed glycosaminoglycans, and manganese ascorbate for the purpose of promoting glycosaminoglycan production. Orally administered glucosamine sulfate has been associated with relief of clinical signs of DJD and chondroprotection in clinical and experimental studies in man, horse and dog. Although glucosamine has a slower onset of relief of clinical signs associated with DJD as compared to ibuprofen, two clinical trials found it to have equal long term efficacy. No significant side effects have been reported with Cosequin.

Diagnosis and Treatment of Rear Limb Disorders

Don Hulse DVM Diplomate ACVS, ECVS

Hip dysplasia is an abnormal development of the coxofemoral joint. The syndrome is characterized by subluxation or complete luxation of the femoral head in the younger patient while in the older patient mild to severe degenerative joint disease is present. Laxity in the hip joint is responsible for the early clinical signs and joint changes. Subluxation stretches the fibrous joint capsule, producing pain and lameness. When the surface area of articulation is decreased, this concentrates the stress of weight bearing over a small area through the hip joint. Subsequently, fractures of the trabecular cancellous bone of the acetabulum can occur, causing pain and lameness. The cancellous bone of the acetabulum is easily deformed by the continual dorsal subluxation of the femoral head. This piston-like action causes a wearing of the acetabular articular surface from a horizontal plane to a more vertical plane causing subluxation to worsen. The physiologic response to joint laxity is proliferative fibroplasia of the joint capsule and increased thickness of the trabecular bone. This relieves the pain associated with capsular sprain and trabecular fractures. However, the surface area of articulation is still decreased causing premature wear of articular cartilage, exposure of subchondral pain fibers and lameness. This may occur early in the pathologic process or later in life. There are two general recognizable clinical syndromes associated with hip dysplasia: (1) patients 5 to 16 months of age, (2) patients with chronic degenerative joint disease. Patients in group 1 present with lameness between 5 to 8 months of age. Symptoms include difficulty when rising after periods of rest, exercise intolerance, restlessness at night, and intermittent or continual lameness. The majority of young patients will spontaneously improve clinically around 15 to 18 months of age. This clinical improvement is due to pain relief as proliferative fibrous tissue prevents further capsular sprain, and increased thickness of the subchondral bone prevents trabecular fractures. If symptoms occur later in life, they may include difficulty in rising, exercise intolerance, lameness following exercise, atrophy of the pelvic muscle mass, and a waddling gait with the rear quarters. Physical findings in the younger group of patients include pain during external rotation and abduction of the hip joint, poorly developed pelvic muscle mass, and exercise intolerance. Hip exam performed under general anesthesia will reveal abnormal angles of reduction and subluxation reflecting excessive joint laxity. Physical findings in the older group of patients include pain during extension of the hip joint, reduced range of motion, atrophy of the pelvic musculature, and exercise intolerance. Radio graphically, there are seven grades of variation in the congruity between the femoral head and acetabulum established by the Orthopedic Foundation for Animals. Excellent, good, fair, and near normal are considered within a range of normal. Dysplastic animals fall into the categories of mild, moderate, and severe. It is important to note that clinical signs do not always correlate with radiographic findings. Recently, patients have been evaluated using a distraction index where the degree of hyperlaxity is measured and correlated with standards for each breed.

Treatment is dependent upon the age of the patient, the degree of patient discomfort, physical and radiographic findings, client expectations of patient performance, and financial capability of the client. Conservative treatment is beneficial to a large number of patients in both the young and older patient groups. Conservative management is divided into acute management and long term management. When a dog exhibiting signs of hip dysplasia enters the clinic, it is generally because they have sprained the hip joint. The dysplastic joint is either hyperlax (young dog) has a limited range of motion (mature dog). In either case, the joint is easily sprained and the dog that is presented with symptoms has generally overused (sprained) the hip joint. The

management of the case at this time period is the same as treating any other acute sprain. Rest, physical therapy, and non-steroidal analgesics will relieve signs in the majority of patients. Rest is just that!!!, controlled activity with slow walking on a leash only. There should be NO free activity for 2 weeks. Physical therapy includes cold therapy for the initial 1-4 days. Commercial cold packs are the most convenient and precise way to apply cold therapy. The application of cold should only be 5-10 minutes. The attending veterinarian must emphasize that REST and PT are the most important considerations when treating an acute sprains.

Following the acute phase of treatment, the attending veterinarian must consult with the owner regarding longterm management of the dysplastic dog. The foundation for long term management of any arthritic joint is weight control, exercise therapy, and anti-inflammatory drugs or supplements. The majority of mature dogs with hip discomfort are over weight. Studies have shown a significant improvement in function if an ideal target weight is achieved. The foundation for weight control is exercise therapy, diet, and owner behavior modification. Administration of drugs (NSAIDs, steroids, PSGAGs, Hyaluronate) or supplements (glucosamine, chondroitin sulfate, manganese) are useful to control discomfort. This is particularly true in the early stages of treatment before the benefits of weight reduction and exercise therapy are realized. The administration of drugs should be at a minimum level (dose and frequency) to achieve comfort. Supplements of glucosamine, chondroitin sulfate and manganese alone or in combination have been shown in vitro as well as in clinical studies to ameliorate discomfort or reduce the dose of drugs needed to control discomfort.

Surgical intervention also is divided into techniques useful in the younger population and those useful in mature dogs. Techniques useful in the younger population include Triple Pelvic Osteotomy (TPO), Double Pelvic Osteotomy, femoral head ostectomy, and possibly total hip replacement. My preference in this aged dog is either a TPO or DPO. The advantage of DPO is that the floor of the pelvic canal is stable is that the ischium does not undergo an osteotomy as in a TPO. This concept allows for greater patient comfort and therefore, the ability to perform a bilateral DPO at the same setting. This reduces postoperative rehabilitation time and allows more rapid return to function. Pelvic osteotomy is used in the group of younger patients to axially rotate and lateralize the acetabulum in an effort to increase dorsal coverage of the femoral head. This procedure is indicated in patients that will lead athletic lives such as the working breeds or in those patients in which the client wishes to arrest or slow the progress of osteoarthritis associated with hip dysplasia. The most favorable prognosis is in patients having minimal existing radiographic degenerative changes and an angle of reduction less than 45 degrees and angle of subluxation less than 15 degrees. The prognosis is less favorable in patients with existing degenerative changes and angles of reduction and subluxation greater than those given above. The details of the technique are beyond the scope of this handout. Briefly, the degree of axial rotation of the acetabulum is set by the previously determined angles of reduction and subluxation. The angle of reduction is the maximum degree of rotation and the angle of subluxation is the minimum degree of rotation. The most commonly used angle of acetabular axial rotation is slightly less than the measured angle of reduction. The pelvis is cut through the pubic brim and body of the ilium. The acetabulum is rotated axially, lateralized and stabilized with the appropriate osteotomy plate. The use of locking technology is an advantage that has decreased post operative implant failure. Postoperatively the patient is restricted to exercise on a leash only until radiographic healing of the osteotomies is complete



Pre Op



Post op DPO



6 weeks

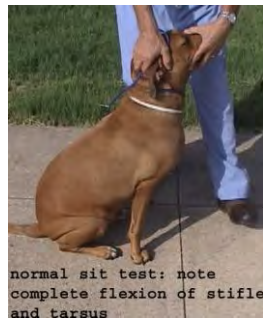
In the older dogs, my preference is total hip replacement or conservative management. Femoral head ostectomy is an option in cases where conservative management is no longer effective and financial constraints precludes Total Hip Replacement. Advancement in Total Hip Replacement is the advent of cementless systems. Cementless systems have decreased the incidence of acetabular cup loosening and femoral stem loosening. Hybrid insertion with cementless cup and cemented stem are often used in dogs with “stove pipe”, ie, uniform diameter marrow cavity.



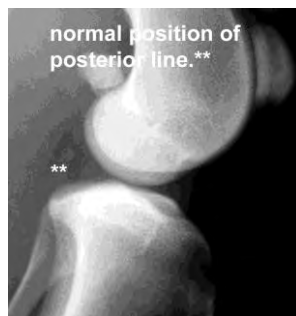
Diagnosis of the CCL Deficient Stifle

Examination: Perform the initial examination of the stifle with the animal standing. Simultaneously palpate both stifles to detect swelling. A swollen stifle usually indicates degenerative joint disease. The patellar ligament becomes less distinct with joint effusion and the medial aspect of the stifle enlarges because of capsular thickening and osteophyte formation. Palpate the stability of the patella with the hip joint in full extension.

Ask the animal to sit; observe the flexion of the stifle and tarsus. The earliest sign of stifle joint pathology is failure to dorsiflex the tarsus fully (compare to the opposite normal side).

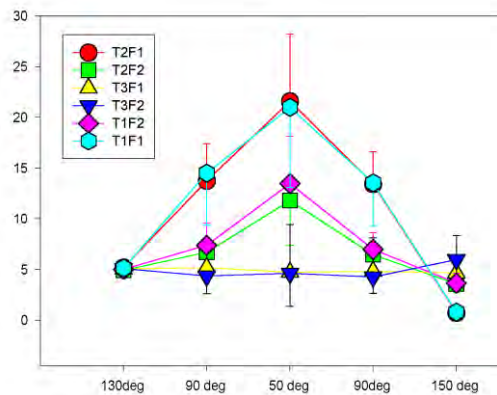


Imaging: Early diagnosis is dependent upon radiographic presence of joint effusion. A radiolucent line adjacent to the caudal joint capsule is representative of fatty tissue in the space between the joint capsule and popliteal muscle. Caudal displacement of this line is representative of joint effusion. This is one of the earliest radiographic indications of partial anterior cruciate ligament injury. As changes progress, typical radiographic signs of DJD will be noted.

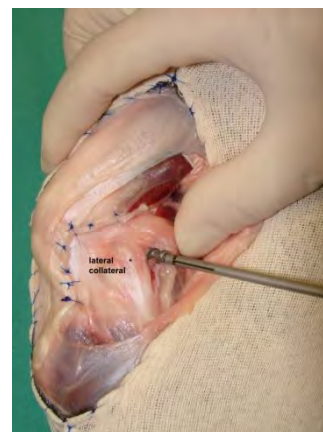
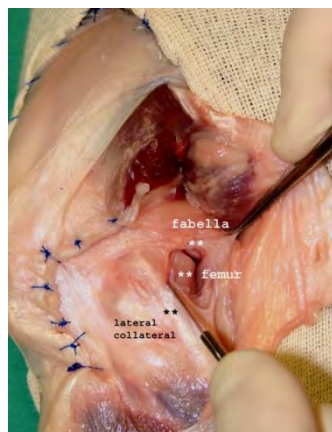


Stabilization of the CCL deficient joint can be accomplished through a variety of methods. Surgical techniques have been developed including placement of intra-articular grafts, insertion of suture material and/or advancement of periarticular structures outside the joint (extracapsular), and tibial osteotomies that alter joint mechanics. The technique of choice is based on surgeon experience. Tibial plateau leveling techniques are preferred by the author in large athletic breeds, with early partial CCL injury, and in dogs/cats with excessive slope. Recent double blind study showed that in larger breeds of dogs, the Nylon crimp technique was not as effective in all outcome parameters as a leveling osteotomy. A number of reasons why the nylon / crimp technique is ineffective have been elucidated. The placement of the nylon (attachment sites at the femur/tibia) are very non-isometric and predispose to suture elongation/breakage. The nylon material itself undergoes stress relaxation/creep, ie, elongates under continual load. Newer materials (Arthrex FiberWire/Tape) have improved structural/mechanical properties. More isometric sites have been identified; note the ligament is very complex and there is no true one isometric site. Studies in our lab at TAMU have identified near isometric sites. A discussion of the site(s) location and technique of application are presented below. Additionally, a leveling osteotomy technique is described for those who wish to apply this method based upon personal preference/indications.

Recommended sites for isometric suture placement:

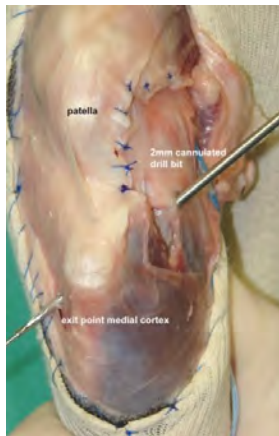
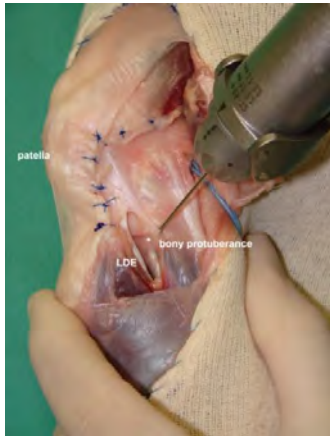


Locating the F2 site: The F2 site is located at the level of the distal pole of the fabella. Placement of the anchor is critical. The anchor must be placed in the femoral condyle as far distal and as far caudal as is possible. An anchor placed too far proximal or anterior is at risk for pull out or suture failure. To locate the correct placement site in the femoral condyle, palpate the distal pole of the fabella. Make a vertical incision through the capsular tissue to expose the joint line between the fabella and caudal margin of the femur. Locate the proper position for the anchor just distal to the fabella-femoral joint line and as far caudal as possible. A hole is pre-drilled with a 2mm drill bit (or 1/16 Steinmann pin) at the correct anchor position. The drill hole is angled directed toward the patella to cranial to eliminate the risk of entering the joint. Insert the appropriate size anchor.

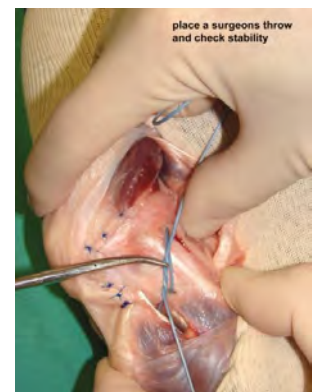


Locate the T3 site at the proximal tibia. First locate the protuberances cranial and caudal to the long digital extensor groove. Make a vertical incision through the capsular tissue overlying the extensor groove. Palpate and locate the protuberance just caudal to the extensor groove; this is the site for placement of the first drill hole. At this site beginning as proximal as is possible without entering the joint, insert a .045 k-wire. The K-wire is directed to glide beneath the extensor groove to exit through the medial cortex of the proximal tibia. With the K-wire in

place, place a 2mm cannulated drill bit over the wire to create the first drill hole. Drill over the K-wire to exit through the medial cortex. Leave the drill bit in place and remove the K-wire. Through the cannulated hole in the drill bit, place a nytilol Arthrex suture passer such that the loop is lateral. Remove the drill bit and leave the suture passer in the drill hole.



Passing the suture through the drill hole: Place one of the suture ends through the loop in the nytilol suture passer. Only place about 1cm of the suture through the loop to decrease suture drag as it passes through the drill hole. Pull the suture passer medial such that the free end of the suture exits through the medial cortex. Place the free end of the suture through a two hole button such that the button will lie against the medial cortex when the suture is pulled taught. Re-insert the nytilol suture passer through the drill hole such that the loop is positioned medial. Place the free end of the suture through the nytilol loop (1cm of suture end) and pull the suture laterally. Now both free ends of the suture are lateral and ready to be tied.



Tying the suture: Place the limb in normal standing position (140 degrees). Place the initial double throw of a surgeons knot and check cranial drawer. Do not over constrain; there should be 2-3mm cranial translation. When satisfied with stability, complete the surgeons knot and place 4 additional half throws. Check range of motion and cranial drawer.

Knotless Swivelock

The 5.5mm PEEK SwiveLock is recommended for dogs weighing 50lbs or greater. One strand of 2mm Fibertape (2 limbs) is inserted for dogs up to 70 lbs or so; two strands (4 limbs) of 2mm fiberTape is recommended for dogs greater than 70 lbs.

The F2 and T3 sites described previously are used in this application.



Concept of CORA Based Leveling Osteotomy

Recent studies have shown joint mechanical alteration that may be contributory to articular cartilage lesions noted on 2nd look arthroscopy. One explanation for reported abnormal joint mechanics with Tibial Plateau Leveling Osteotomy is that the standard Slocum osteotomy is not based on the anatomic CORA. As such, the Axis of Correction (ACA) is not aligned with the anatomic CORA resulting in mal-alignment of the proximal and distal anatomic axis and secondary translation. The result is caudal displacement of the weight bearing axis and a focal increase in joint force. When rotated to the recommended 5 degrees, the long-term effect is loss of compliance of cranial supporting structures such as the fat pad and joint capsule. Encroachment of the cranial supporting structures (joint capsule) on the cranial articular surface of the medial/lateral femoral condyles can result in abrasion of the articular cartilage.

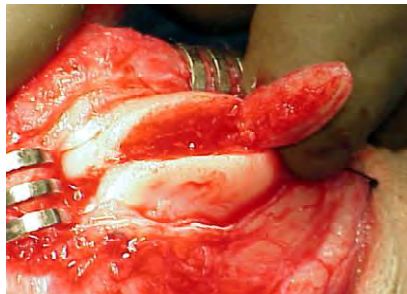


Surgical Techniques for Stabilization of the Patella

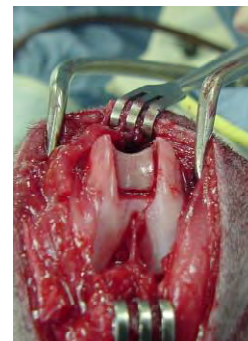
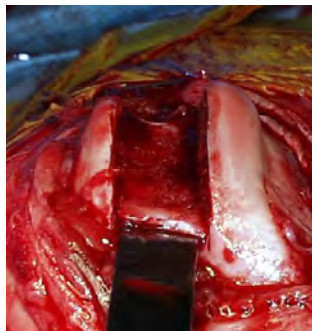
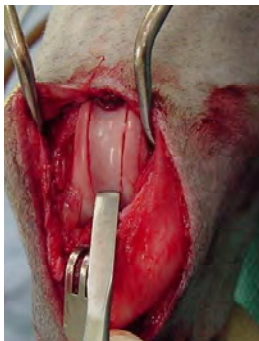
Deepening of the Trochlear Groove.

If the medial and lateral trochlear ridges do not constrain the patella, the trochlear groove must be deepened. This technique is generally necessary in dogs/cats with a grade III or IV luxation. Deepening the groove may be achieved with a trochlear wedge recession, trochlear block recession, or trochlear resection.

Trochlear wedge recession deepens the trochlear groove to restrain the patella and maintains the integrity of the patellofemoral articulation. Make a diamond shaped outline cut into the articular cartilage of the trochlea with a scalpel (use smooth arcs rather than corners on the lateral and medial sides of the diamond). The width of the cut must be sufficient at its' midpoint to accommodate the width of the patella. An osteochondral wedge of bone and cartilage is removed by following the outline previously made. Make the osteotomy so that the two oblique planes that form the free wedge intersect distally at the intercondylar notch and proximally at the dorsal edge of the trochlear articular cartilage. Use caution to avoid making the wedge too long (may affect cruciate ligament insertions) or too deep (may go through the caudal aspect of the femur).



Trochlear block recession is performed similarly to the wedge recession. Some surgeons find the block recession most appropriate for dogs that seem to luxate primarily with the stifle joint in extension, and for larger breed dogs. The advantage of the block recession is deeper placement of the patella and more advantageous proximal tracking of the patella into the trochlear groove.



Tibial tuberosity transposition: Tibial tuberosity or crest transposition is an effective method of treatment for grades II, III, and IV patellar luxations. **Medial release:** The medial joint capsule is thicker than normal and contracted in patients with grade III or Grade IV patella luxations. In this group of animals, the medial joint capsule and retinaculum must be released to allow lateral placement of the patella.

The pull of the sartorius muscles and vastus medialis muscle directs the patella medially, the insertions of these muscles at the proximal patella are released. Redirect the insertions and suture them to the vastus intermedius.



Lateral reinforcement: Reinforcement of the lateral retinaculum is accomplished with suture placement and imbrication of the fibrous joint capsule, by placement of a fascia lata graft from the fabella to the parapatellar fibrocartilage, patella sling suture, or excision of redundant retinaculum.

Femoral varus/valgus correction is required in cases where the angulation of the distal femur precludes correct alignment of the extensor mechanism. This abnormal / excessive varus/valgus is present with cases of Grade 4 patella luxation. The method of planning correction is based upon the CORA methodology. CT imaging and reconstruction are the most accurate method of determining correction. In severe cases, prototype development is ideal. Many centers do not have the ability to perform CT/reconstruction and prototype development; accurate radiographic positioning and planning will suffice except in the more severe cases. These cases can be very complex and therefore recommended only for the experienced surgeon.



Decision Making in Fracture Management

Don Hulse DVM, ACVS, ECVS

Basic Principles

- A) Fracture fixation and healing is essentially a “race” between failure of the implant system utilized to stabilize the fracture and the organized process of wound healing and fracture repair.
- B) Although a thorough knowledge of regional anatomy, meticulous aseptic technique, respectful tissue handling, and understanding of orthopedic implant biomechanics are pre-requisites for successful fracture fixation, **the majority of fracture fixation failures occur because of poor decision making which occurs prior to entering the surgical suite.**
- C) Why are orthopedists prone to poor decision making when treating fractures?
 1. **Radiographs** are the standard of care diagnostic tool for most fracture patients. This imaging modality is mandatory for proper diagnosis and classification of fractures. Moreover, radiographic interpretation partially (note I did not state completely) dictates the fixation system that can be selected for fracture repair. Thus, early in the course of **treating a patient** for a fracture, the orthopedist begins to think in the terms of the affected bone as it is represented on a radiograph, and not of the injured bone as part of an injured patient.
 2. The problem with this mindset is that focusing solely on the fractured bone often leads the orthopedist to ignore the supporting tissues around that bone and the clinical factors of the patient. **Although is commonly said that orthopedists are nothing more than “frustrated carpenters”, a fractured bone is not just an inanimate biomaterial desperately in need of plates, pins, and screws. An injured bone should be thought of as an injured organ consisting of a variety of hard and soft tissues that need to be properly treated for timely healing to occur.** Without a healthy, robust supporting soft tissue envelope, a fractured bone will not heal. Adequate blood supply, intact musculotendinous attachments, and patient comfort leading to early return of limb use are all mandatory for fracture healing and a clinically successful outcome. **Failure to: 1) asses the health of these soft tissues, 2) respect them during surgical approach to the fracture, and 3) protect them during implant placement WILL RESULT in inadequate fracture healing and subsequent failure of the applied fixation device.**
 3. The third element often resulting in poor fracture decision-making involves failure to consider the veterinary-client-patient relationship. The veterinary orthopedist can make the most accurate assessment and fixation plan for an injured bone and its surrounding soft tissues, **but if he/she fails to consider the client that will be caring for the injured veterinary patient, the chances for post-operative complications and subsequent failure of the fracture repair is markedly increased.**

D) **Thus, the basic principles for proper fracture decision-making are to consider three factors independently for every patient: mechanical factors, biologic factors, and clinical factors (client constraints).** In fact, a numerical score, referred to as a **Fracture Assessment Score (FAS)**, should be generated for each of these 3 factors. After scoring each factor, an overall “average” score is then generated for the patient. This patient’s FAS is then used to guide choice of fixation systems.

2) **More on the Fracture-Assessment Score: *Why is it important?***

A) Early, progressive consolidation of a fracture during the post-operative period causes a shift in relative load bearing toward the healing bone and away from orthopedic implants. Conversely, slower fracture healing dictates that the fracture fixation system provides stability longer, which increases the likelihood of implant-related complications. Thus, each fracture repair truly represents a “race” between implant failure and fracture healing. As stated above, poor decision-making occurs for a variety of reasons, such as observation of the fracture on radiographs or comparing how a fractured bone appears in relation to a surgical textbook diagram. Recurrent fixation failure due to poor decision-making leads surgeons (and often their clients!) to become frustrated with orthopedic case management. This frustration often occurs because of poor decision-making, and not because of the surgeon’s abilities.

3) **The fracture-assessment score (FAS) ranges from 1 to 10.** The lower end of the scale represents mechanical, biologic, and clinical factors that do not favor rapid bone union and return to function, while the upper end of the scale represents those factors that favor rapid bone union and return to function.

A) Specific Factors:

1. Mechanical Factors

(1) Number of limbs injured:

(a) Dogs and cats must bear weight on 3 limbs; weight bearing on the implant-bone construct cannot be prevented post-op when multiple limbs are injured or when pre-existing conditions exist (severe OA secondary to chronic CrCL rupture, limb missing from previous amputation).

(2) Patient size and activity:

(a) Complication rate is directly related to the stresses applied to implants during the post-operative recovery period. Excessively loaded implants have an increased rate of failure. Large/active patients subject implants to greater loads and are more prone to have implants loosen prematurely and/or fail.

(3) Ability to achieve load-sharing fixation between the bone and the implant:

(a) Ideal implant-bone load sharing occurs when a transverse or short oblique fracture is repaired using **DIRECT REDUCTION TECHNIQUES**, because restoration of the bone column allows loads

to be transmitted axially through the limb (and not solely upon the implant). In these situations loading of the implant is minimized and implant loosening/failure is less likely.

- (b) Conversely, implant-bone load sharing does not occur when **INDIRECT REDUCTION TECHNIQUES** are utilized, because loads are transmitted from bone segment to bone segment COMPLETELY through the selected implants [as in a highly comminuted fracture that cannot (and should not) be anatomically reconstructed].
 - (i) **This does not mean that indirect reduction is inferior to direct reduction!** In fact, indirect reduction techniques have a number of advantages over direct reduction techniques. However, if indirect reduction is selected, the implant(s) must be properly sized and positioned to absorb loads throughout the healing process.

2. Biologic Factors

(1) Patient age:

- (a) Young dogs and cats (< 6 months) are considered “healing machines” and require less robust fixation devices and these devices are typically maintained for a reduced amount of time. Conversely, geriatric patients demonstrate delayed healing, which subjects implants to increased number of cycles and an increased incidence of implant failure.

(2) Open vs. closed fracture:

- (a) Open fractures are associated with a significant degree of soft tissue injury, resulting in longer time to bone union because soft tissues must heal first. Closed fractures tend to have less soft tissue injury and in general heal more quickly.
- (b) In addition, open fractures are often not ideal candidates for fixation systems that will remain with the patient long-term (bone plates, interlocking nail, cerclage wire), as the application of large, non-absorbable metallic implants into a contaminated environment may result in complications such as implant infection, incisional dehiscence, long-term draining tracks, and implant failure.

(3) Low vs. high energy injury:

- (a) Low energy fractures (fall from the bed, stepped on by client) have less soft tissue injury when compared to high-energy fractures (gunshot wounds, high-speed hit by car), which are often comminuted in nature. *****One way to think about “energy” is to consider the fracture configuration.** Simple, two-piece fractures are typically low energy, whereas highly comminuted fractures are high-energy. If a high-energy injury was applied to the bone, that same level of injury traumatized the surrounding soft tissues.

(4) Choice of open vs. closed reduction:

- (a) Open reduction techniques, by their nature, damage supporting soft tissues. Conversely, closed reduction techniques such as external coaptation or closed application of an external skeletal fixator or IM pin result in minimal iatrogenic soft tissue injury. **One of the MOST POWERFUL biologic influences is the surgeon's skill in minimizing soft tissue damage during open reduction.** Obtaining the desired fracture fragment reduction and fracture stability while causing minimal soft tissue injury in the shortest possible operative time results in the greatest success. **Thus preservation of soft tissues during open approaches is of the UTMOST importance.**
- (b) This concept has led to a number of more "biologically friendly" fracture fixation techniques referred to as *bridging osteosynthesis, or biologic osteosynthesis*. Examples of these techniques include:
 - (i) Open-but-don't-touch (OBDT) - use of a standard open approach without disturbing the fracture hematoma or the fracture fragments
 - (ii) Mini-approach to the fracture for reduction with closed application of external skeletal fixation – mini-approach utilized to visualize fracture and achieve reduction, but fixation system applied in a closed manner.
 - (iii) Minimally invasive plate osteosynthesis (MIPO) - creating small "portals" over parent bone fragments for placement of implants, followed by sliding the implants from one portal to the other beneath an intact segment of skin and soft tissue

(5) Amount of surrounding soft tissue:

- (a) Bones with little surrounding soft tissues, such as the radius/ulna and distal tibia result in delayed unions and complications more often than similar fractures affecting the humerus, femur, or pelvis. *****The poster-child for this phenomenon in small animal orthopedics is the distal radius/ulna fracture in toy and small breed dogs. Failure to recognize this patient population prior to initiation of treatment WILL RESULT in delayed or non-unions and a number of severe complications.**

(6) Location on the bone:

- (a) Fractures of the diaphyseal bone heal more slowly and are inherently less stable after reduction because of the reduced blood supply and limited surface area of fracture ends. Fractures involving the metaphysis of bone heal more quickly because of improved blood supply and greater surface area for contact between fracture ends.

(7) Concurrent diseases or medications:

- (a) Diseases that delay healing must be considered when choosing fixation systems
 - (i) Hypothyroidism
 - (ii) Hyperadrenocorticism
 - (iii) Iatrogenic corticosteroid or chemotherapy treatment
- (b) Conditions that increase wound infection rates must also be considered when selecting internal vs. external fixation systems

- (i) Hyperadrenocorticism
- (ii) Iatrogenic corticosteroid or chemotherapy
- (iii) Diabetes Mellitus (little evidence to support this as a risk factor in veterinary orthopedic patients assuming the patient is well regulated)

3. Clinical Factors

(1) Client's willingness/ability to participate in aftercare:

- (a) A client who works 80-100 hours/week and commutes 2 hours a day is probably not the best client to task with daily external fixation pin-tract care or weekly re-check visits, whereas a retired individual or someone who works from home may be well suited for this added time commitment.

(2) "The bigger they are, the harder they fall!"

- (a) Assuming your client has the intestinal fortitude to provide certain post-operative treatments is fraught with danger. Some of the toughest looking clients end up on the exam room floor the first time they see an external fixator attached to their beloved pet, while meek/timid clients often handle this situation well. Be certain to counsel each client on the proposed fixation system PRIOR to fracture repair.

(3) Compliance with post-operative exercise restriction:

- (a) A client who is unlikely to confine the patient post-operatively often requires selection of more robust implants in order to cope with the increased load and cycling that will be placed on these implants if confinement instructions are not followed. Additionally, dogs or cats with clients that are unlikely to comply with post-op instructions should be treated with internal fixation systems, as external fixators and casts/splints often lead to complications in this scenario.

(4) Patient's ability to cope with discomfort:

- (a) Each individual patient's "stoic nature" must be closely considered. For example, a patient from a stoic breed such as a Staffordshire terrier will often ignore external skeletal fixation devices and walk on the injured limb as if it was un-injured. Application of a similar device to a patient from a "wimpy" breed such as a Toy Poodle or Maltese will result in complete refusal to use the limb. Refusal to bear weight on the injured limb results in delayed union, disuse osteopenia, and non-union or stress-protection related fractures.

4) Put it all together: The patient's overall Fracture-Assessment Score (FAS)

A) As stated, mechanical, biologic, and clinical factors should be individually considered and scored. These 3 scores are then combined, or averaged, to provide a whole patient FAS.

1. Low Scores (0-3)

- (1) In this category, implants often bridge large fracture gaps and therefore must have sufficient strength to prevent permanent bending or breakage. Implants must also deter excessive motion at the implant-bone interface.

Motion at this interface will cause bone resorption, premature implant loosening, and implant migration. Implants should purchase bone with raised threads (bone screws utilized to secure bone plates, positive profile transfixation pins used with external skeletal fixation (ESF)).

- (2) **Suggested implants** include lengthening plates, bone plate/IM rod combinations, bone plate/ESF combinations, type II or III ESF, ESF/IM pin combinations, and interlocking nail fixation.

2. Intermediate Scores (4-7)

- (1) Overlapping biologic and mechanical factors affect healing and implant selection in this category. If the implant and bone share the load following surgery, the implant will be subjected to less stress; however, healing may still be delayed due to a poor biologic score. Alternatively, biologic score may be excellent, but the implant may be subjected to high initial loads because it is buttressing (bridging) the fracture. A score toward low-center means that time to union will be long and thus the implant must purchase the bone in a way that will preserve implant-bone interface. A score toward high-center means that the stress on the implant and implant-bone interface will be high for a short period of time, thus the implant-bone interface needs maximum stability until callus formation occurs (which reduces implant stress).

- (2) **Suggested implants** include bone plates, Type I or II ESF, IM pin/ESF combinations, interlocking nail fixation, and rarely IM pin/cerclage wire combinations.

3. High Scores (8-10)

- (1) When the FAS is greater than 7, immediate load sharing between the bone-implant construct and rapid bone union is expected. Therefore the strength and stiffness of the fixation system need not be extreme, nor does the implant need to function for a long period of time.

- (2) **Suggested implants** include type I ESF, IM pin/cerclage, and external coaptation. Implants that hold bone through frictional purchase (smooth tipped pin, cerclage wire) often provide adequate bone purchase.

5) **Concluding remarks: Treat the patient, and not the radiograph!**

- A) In conclusion, successful fracture fixation requires a thorough knowledge of regional anatomy, meticulous aseptic technique, respectful tissue handling, and understanding of orthopedic implant biomechanics. However, the single most important factor in successful fracture treatment is proper decision making **PRIOR** to incision of skin. This is best summarized by the phrase “**treat the patient, not the radiograph**”. After careful consideration of mechanical, biologic, and clinical factors, a patient FAS is generated. The FAS is designed to properly match the selected fracture fixation system with each patient. By considering all of the factors that result in the FAS, proper selection of fixation systems and their method of application will dramatically reduce post-operative complications and fracture fixation failure.

Intra-medullary Pins & Ancillary Stabilization Devices

Introduction

Intramedullary (IM) pins and cerclage wires are relatively easy to apply, do not require expensive equipment and, when used appropriately, can be very useful for managing many types of basic fractures in dogs and cats. IM pins and cerclage wires have gained a bad reputation, as they can be disastrous when used inappropriately. A fundamental understanding of fracture biomechanics, implant biomechanics, and fracture decision making are necessary in order for the surgeon to determine whether or not pins and wires are appropriate for individual patients.

IM Pins

Intramedullary pins (IM pins) used in animals range from ¼ inch diameter (6.3 mm) down to 5/64 inch diameter (2.0 mm). Intramedullary pins in this size range are called Steinmann pins. Smaller pins are usually referred to as Kirschner wires (K-wires), and although they may be used as intramedullary devices in very tiny animals, they are generally used as inter-fragmentary devices. K-wires are available in .035, 0.045, .054 and .062 inch diameters. Intramedullary pins and K-wires can be obtained as fully threaded, partially threaded or nonthreaded. Although partially (end) threaded pins are used as intramedullary pins with the intention of increasing rotational stability, those pins do not provide additional stability and are at risk for breakage at the thread-shaft interface. In addition, partially threaded Steinmann pins and K-wires are more difficult to remove as the bone tends to grow into the threads. *For these reasons, the use of threaded pins as intramedullary devices is not recommended.*

Steinmann pins and Kirschner wires are available in a variety of lengths, usually from 6-12 inches long, and may have points on one or both ends. The pins are easily cut, and there is no advantage to the veterinary surgeon in purchasing single pointed pins. Pins may be manufactured with a trocar or chisel/diamond point. *Trochar points* are by far the most commonly used and consists of a three-sided tip with a long bevel and good ability to penetrate cortical bone. *Chisel points (also called diamond points)* are broad, flat two-sided points with a short bevel and are designed to deflect the pin away from the cortex during drilling rather than engage the opposite cortex.

Intramedullary pins excel in resisting bending forces in 360 degrees, can be placed without specialized equipment, and often can be placed with a limited approach. Intramedullary pin placement, unless a very large pin is placed or reaming of the medullary cavity is performed, has limited impact on the intramedullary blood supply. Intramedullary pins are relatively easy to remove, in contrast to fixation devices such as screws, plates, or interlocking nails.

Intramedullary pins do not resist rotation or axial (compression/tension) forces, and therefore are rarely used alone but combined with other types of fixation, for example cerclage wires, external fixators, or bone plates. From a mechanical standpoint, use of the largest pin possible will result in the stiffest construct and most resistance to bending. However, use of an excessively large pin has several disadvantages: difficult placement in a curved bone, for example the canine tibia and femur, damage to the intramedullary blood supply, and risk of creating additional fractures if the pin exceeds the diameter of the bone at its' narrowest point, or isthmus. *In general, a pin that is approximately 70% of the diameter of the long bone at the isthmus is chosen.* If the surgeon anticipates combining the pin with another type of device, a

slightly smaller intramedullary pin is chosen. Use of a pin that is too small may result in failure by pin bending or breakage. Use of multiple small pins to fill the medullary cavity, also called “stack pinning” to increase resistance to rotational stability, has been shown to have no effect on rotational stability when compared to a single IM pin.

K-wires, while they can be used as intramedullary devices, are usually used as interfragmentary devices, often to maintain temporary fracture reduction while the primary fixation is applied. K-wires by themselves are relatively weak implants and are typically not used alone. In certain fractures, for example physeal fractures in small dogs and cats, cross-pinning with K-wires or small Steinmann pins can be sufficient when fracture healing is expected to be rapid. K-wires are also commonly utilized in combination with cerclage wire for tension-band fixations and to support full cerclage wires in areas of changing bone diameter (“skewer wires”).

Application Techniques for Intramedullary Pins and Interfragmentary Wires

Intramedullary pins may be inserted either from the fracture site (*retrograde insertion*) or from either the proximal or distal end of the bone itself (*anterior insertion*). The local anatomy of the bone often dictates how the pin is driven, for example, retrograde pin insertion in the tibia from a distal to proximal manner may result in damage to the articular cartilage or cruciate ligaments. An estimation of appropriate pin size (60-75%) may be made from the pre-operative radiographs and may be confirmed by observation of the pin as it is gently introduced into the fracture site if the fracture is treated with an open approach. When in doubt regarding pin diameter, a smaller pin should be used initially and replaced with a larger pin if necessary. The pin may be inserted either open or closed. Although closed pinning, based on palpation, can be performed by the experienced surgeon this can become more difficult in larger animals with soft tissue swelling, or in fractures greater than 72 hours old. The increased use of intraoperative imaging (fluoroscopy) can greatly facilitate IM pin placement in a minimally invasive fashion, with less damage to the soft tissues.

Intramedullary pins may be placed either by hand, using a Jacobs’ chuck, or with a drill. *Hand insertion* may allow the surgeon to feel whether or not the pin is advancing down the medullary cavity and whether it is up against or about to penetrate cortical bone. When placing pins by hand with a Jacobs’ chuck, the chuck should be “choked-up” as far as possible on the pin in order to prevent pin bending during cortical penetration, particularly when smaller pins are used. The chuck should be firmly tightened with the key in at least two separate places to avoid sudden loosening during pin advancement. Jacobs’ chucks are sold with protective devices that are designed to protect the surgeon’s hand from inadvertent pin penetration resulting from sudden pin loosening. It is strongly recommended that this protective device be utilized early in the learning curve. The tip of the pin should firmly engage bone, and the pin rotated back and forth with quarter turns (rather than driven consistently clockwise or counterclockwise). In larger animals with hard cortical bone, hand placement can be very difficult. In addition, the smaller the pin the more difficult it is to drill by hand, thus power insertion is suggested for interfragmentary K-wires.

When using a drill to insert an intramedullary pin or K-wire, a cannulated drill should be used to drive the pin to decrease the amount of pin bending or breakage during drilling. Ideally a high-torque, low speed drill should be used (as opposed to a high speed drill) to decrease heat

generation and subsequent bone necrosis. Saline lavage is also suggested during pin placement to decrease heating of the bone, particularly with K-wire insertion. *When driving an intramedullary pin, whether by hand or with a drill, it is critical to align the pin with the shaft of the bone in both craniocaudal and mediolateral planes.* With open reduction techniques, the surgeon may find it easiest to drive the pin with one hand and stabilize the fracture segment using a bone-holding forcep in the non-dominant hand. Having an assistant hold a second pin parallel to the shaft of the long bone may be helpful, or in certain cases, placing an “aiming pin” retrograde just a few cm into the medullary cavity so the surgeon can attempt to drive the normograde pin along the axis provided by the aiming pin. When attempting to seat an IM pin into metaphyseal bone in the distal fracture fragment, it is important not to penetrate the subchondral bone plate and cartilage. This untoward event is particularly common when placing IM pins in the femur using normograde technique. The surgeon will note an increase in resistance as the pin begins to seat into the metaphysis. In addition, the fracture fragments may begin to distract apart as the pin over-lengthens the bone as it is driven into the metaphysis. In comminuted fractures, an IM pin is an excellent tool to achieve fracture distraction and alignment in both craniocaudal and mediolateral planes. When using IM pins for this purpose, the tip of the pin should be cut as the sharp tip of the pin passes across the fracture. This will allow improved distraction of fracture fragments and prevent penetration of the joint. The distance that the pin is advanced may be judged by using a second pin of equal length and lining the two pins up after the Jacobs chuck or drill has been removed. After the pin has been seated, the proximal and distal joints should be put through a complete range of motion, as it is easy to inadvertently place a pin into a joint. Immediately prior to wound closure, the surgeon should carefully palpate the soft tissues surrounding the bone for evidence of overly long or misplaced pins, which can be difficult to feel as they unexpectedly exit cortical bone during drilling.

After the pin is determined to be in the correct position, it is cut using specialized pin cutters. Pin cutters are generally designed only to cut pins of a certain range in diameter, and inadvertent use of small pin cutters to cut a large pin may result in permanent damage to an expensive piece of equipment. The surgeon should check the range of diameters listed on the side of the cutter. It is helpful to have an autoclavable pin guide in the pack to determine the size of the pin in surgery. Pins accumulate and release substantial energy when cut and have the potential to cause serious injury to the surgeon, assistant or circulating technicians if the free end is not firmly grasped or covered with a sponge or surgical towel when cut. The fracture should be carefully observed during and after pin cutting to make sure that alignment is not disrupted.

The decision to cut a pin flush with the surface of a bone or leave the pin long for subsequent implant removal is a decision that varies among surgeons. Leaving pins long can lead to problems with iatrogenic nerve damage (proximal femur), damage to nearby articular cartilage or the patellar tendon (tibia), or soft tissue irritation with subsequent seroma formation or erosion of the tip of the pin through the soft tissues. The alternative to leaving pins long is to cut them short and countersink them, using a countersink and mallet. If countersinking is performed, the pin should be retracted approximately 1 cm, cut as short as possible, and then gently driven back into the bone with a mallet and countersink.

Special Consideration: Cross Pinning Technique

Cross pinning is a technique commonly employed to treat two-piece Salter-Harris (S-H) fractures

in young dogs and cats. Comminuted fractures or distal long-bone fractures in middle-aged or elderly dogs and cats should not be treated with cross pinning. The most common S-H fracture amenable to cross pinning is the distal femoral S-H fracture. However, S-H fractures of the distal humerus, distal radius, and proximal tibia may also be successfully cross-pinned in select cases. Cross pinning simply denotes the use of two or more Steinman pins for stabilization of a fracture. In the case of a distal femoral fracture, the most challenging aspect of this repair is fracture reduction, owing to the small distal fracture fragment and the caudal and proximal displacement of the distal fragment. Most distal femoral fractures are successfully cross pinned via a standard lateral approach to the stifle and distal femur, but combination approaches and tibial tuberosity osteotomy approaches are occasionally employed. After lateral arthrotomy, the distal fracture fragment is identified and carefully grasped with clamshell or point-to-point bone holding forceps. Care must be taken not to injure this small fragment or the articular cartilage with forceps. A small Hohmann retractor, periosteal elevator, or un-loaded scalpel handle can be used to lever the distal fragment into reduction. Placing the stifle in full extension facilitates this maneuver. The tendency is to partially reduce the fracture without correcting excessive procurvatum (femoral condyles rotated caudally). The proximal fracture lines must be identified and anatomically reduced to prevent this problem, and ensure adequate stability and proper limb use after surgery. The distal femoral physis has a “W” shape when viewed on a lateral radiograph. Anatomic reduction of the fracture results in the physis providing substantial stability to the repair. Once the fracture is reduced, Steinmann pins are driven in a distal to proximal direction across the fracture. Pins should be as large as possible without risking iatrogenic fracture. It is critical for the pins to cross each other proximal to the fracture plane, as this provides maximal implant stability. The lateral pin is started just cranial to the long digital extensor tendon sulcus, aimed and driven proximally and medially toward the medial femoral cortex. The pin is driven until the tip begins to exit the medial cortex. A second pin is started on the medial femoral condyle at the same level as the lateral pin, and aimed proximally and laterally to exit the lateral femoral cortex. The pin is also driven until the tip just begins to exit the lateral cortex. Additional pins may be required in challenging cases. Stifle range of motion and fracture reduction are evaluated. Pins are backed out 5-10 mm, cut as flush to the bone as possible, then driven below the surface of the articular cartilage with a mallet and countersink. The ends of the pins must be at or below the level of the cartilage. Routine closure is performed, followed by post-operative radiography. Excessively long pin tips protruding proximally may warrant a return trip to the OR, as these pins are often impinging the quadriceps and cause substantial pain and joint immobility. Instead of opening the primary incision, a mini-approach to the offending pin is often possible and the pin is cut flush with the bone to eliminate post-operative muscle impingement. If possible, a 90-90 flexion bandage, maintained for 2-3 days after surgery, should be considered to maximize stifle range of motion.

Cerclage Wire

Cerclage refers to a wire used to encircle a bone. Cerclage wire typically is used to provide interfragmentary compression in a manner similar to that of interfragmentary screws, and is useful in situations where space is limited and screw application would be difficult, or when screws of the correct type or size are not available. Properly applied cerclage wires do not interfere with the blood supply to bone, and may be used in the immature as well as the mature animal. Types of cerclage wire application include full cerclage, where the wire completely encircles a complete cylinder of bone, and hemicerclage, where wire is passed through a hole or

holes drilled through bone (we will focus on full cerclage wire).

For the vast majority of fractures, cerclage wire is used as adjunctive, rather than primary, fixation. The surgeon should keep in mind that while properly applied cerclage wire is effective when used in appropriate cases, errors in application or case selection can be disastrous.

Cerclage wire is typically applied to long spiral or oblique fractures where the length of the fracture is roughly two and a half to three times the diameter of the bone, and a single cerclage wire should be avoided as it acts as a stress concentrator and become a fulcrum for motion of the fracture fragments. Cerclage wire may be used both as a temporary reduction device, for example, to hold the fracture in reduction while applying a plate, external skeletal fixator or interlocking nail, or as a permanent device, often in combination with one of the above or with an intramedullary pin. An exception to the single cerclage wire rule for long bones may be made if the intent is to prevent a non-displaced fissure fracture from propagating or fracturing further during manipulation of the fracture ends during open reduction.

Cerclage wire is made of relatively soft (usually annealed) 316L stainless steel that is available on spools, in coils or as preformed loops. Sizes of cerclage wire typically used in cats and dogs include 16 to 22 gauge. The larger number denotes a smaller diameter wire. Wire diameter can exponentially increase load to failure (a 50% increase in diameter may increase load to failure by up to 169%), and the largest diameter wire that the surgeon can apply to the bone without technical difficulty is recommended. Method of wire application and type of knot have been extensively described in both the human and veterinary literature. When reviewing the literature, one should be aware that some studies are designed to evaluate cerclage wire used for spinal or tension band applications rather than for long bone applications. The results of spinal stabilization or tension band studies should not be directly applied to long-bone stabilization.

Cerclage Wire Knots and Wire Tensioners

Cerclage wire in veterinary surgery is generally applied either as a twist wire or loop wire (single or double loop). Clinical advantages of twist wires include ease of application with a wire twister (a large instrument that resembles a needle holder but is designed to grasp pins and wire), the ability to tighten and fasten the wire at the same time, and the ability to retighten the wire if it loosens during fracture reduction, as frequently occurs during the placement of multiple cerclage wires. Loop wires have the advantage of better initial tension or tightness when properly applied, and do not have a protruding twisted end that may irritate soft tissues. Loop wires that loosen during fracture reduction must be removed and replaced. There are many types of wire tensioners available or reported in the literature. They fall into categories of twist tighteners, loop tighteners and there are instruments available that can actually tie a square knot in stainless steel cerclage wire. We will focus on a wire tensioner that is used for placement of single or double loop cerclage wires.

Application of Full Cerclage Wire

For fixation of a long bone fracture, cerclage wire should ONLY be used where the fracture can be anatomically reconstructed to complete the original, 360 degree cylinder of bone. Obeying this rule will do more to limit fracture complications than any other rule or guideline. Although cerclage wires are capable of compressing bone fragments, they cannot maintain fragment position in the presence of a cortical defect. Thus, if a tiny piece of bone is missing, the fracture

will collapse as the wire is tightened resulting in a loose wire and loss of fracture reduction. In general, cerclage is reserved for two-piece long oblique or spiral fractures, although occasionally a third piece may be successfully incorporated, but only if the 3rd fragment is a large butterfly fragment with each arm of the fragment spanning two times the diameter of the bone. It is important to minimize dissection and soft tissue trauma to the musculature attached to the bone while applying cerclage wire. Cerclage wire may be passed around the bone either directly or using an instrument such as a cerclage wire passer. Soft tissue inadvertently trapped under the wire will undergo necrosis, subsequently leading to wire loosening. However, there is no need to attempt to place the wires sub-periosteally. Wires are generally spaced at least one to one-half bone diameter's apart from each other, and should always be placed 3 wire diameters away from the proximal or distal most aspects of the fracture fragments.

After the cerclage wire has been carefully passed around the diaphysis of the bone and the fracture reduced, it must be tightened while maintaining reduction. The easiest way to maintain reduction is to place two reduction forceps at right angles to the fracture planes so that both hands may be used for wire placement. For twist wire application, the wire may be twisted by hand for the first one or two twists loosely, leaving about 0.5 to 1 cm between the bone and the twist. A wire twister should be used for applying cerclage wire, as use of an ordinary pair of pliers allows loss of tension as the wire is being twisted. Both wire ends are grasped with the twister where they intersect, and the wire pulled firmly away from the bone while at the same time twisting. This prevents one end of the wire from wrapping around the other, which drastically weakens the wire. *The wire MUST be tight!* The surgeon should watch as the gap between the wire and the bone disappears, and should periodically check the wire for looseness by pushing firmly on it with an instrument. If the wire is loose, tightening should continue. With practice, the operator will develop a "feel" for the mechanics of stainless steel cerclage wire. If you are an inexperienced surgeon and have not broken a few wires during the learning process, you are likely not tightening your cerclage wires appropriately. If the wire breaks between the 2nd and 3rd twist or higher and is tight, it may be left in place, otherwise, it is removed. The wire should be left without cutting or otherwise manipulating the ends until all cerclage wires have been placed, and then checked again for tightness. Care should be taken not to damage the length of wire that is going to stay in the patient, as even a small notch or defect will greatly decrease the fatigue resistance of the wire. Loose wires should either be retightened or removed and replaced. If the wires were placed for temporary fixation, for example to hold the fracture in reduction while applying a bone plate, they may be removed prior to final tightening of the plate screws. Just prior to closure, twist wires should be cut to preserve at least 2-3 twists. It has been shown that wiggling the end of the wire during cutting can substantially decrease the tension in a twist wire. In addition, wire ends should *not* be bent over with twist wires when they are utilized as full cerclage (as opposed to the pin and tension band technique).

Special Consideration: Pin and Tension Band Technique

The pin and tension band technique is uniquely suited to stabilize fractures that receive a distraction force (a muscle or tendon pulling one fragment away from the parent bone) during recovery. The most common fractures that experience this environment are tibia tuberosity fractures, olecranon fractures, greater trochanter fractures, and medial/lateral malleolar fractures of the distal tibia/fibula. While pin and tension band fixation is commonly employed to stabilize surgically induced osteotomies (anconeal process to access the shoulder, olecranon to access the

elbow, greater trochanter to access the hip, acetabulum, ischium), this fixation system is extremely effective for traumatic avulsion fractures of these same bones.

Proper pin and tension band fixation involves reduction of the fracture fragments, temporary stabilization with reduction forceps if possible, and placement of two K-wires or small IM pins across the fracture. The pins should be driven as perpendicular to the fracture plane as possible, and should be started at approximately the same level in the proximodistal direction. The pins are driven toward the opposite cortex until the tip of each pin just begins to exit the far cortex. A hole is next created in a caudocranial direction distal to the fracture. The hole should be 2-3 wire diameters away from the periosteum/cortex, as well as equidistant from the fracture when compared to the pins. A strand of cerclage wire with a small pre-formed loop is next passed through the tunnel, and wrapped around the ends of the IM pins in a figure-of-eight manner. The two free arms of the cerclage wire are connected and a standard wire twist is initiated. This results in a standard wire twist on one side of the figure-of-eight wire, and a second loop within the wire that is used to tension the other side of the figure-of-eight wire. Wire twisters are used to tighten each side of the tension band. Unlike long-bone cerclage wires, the ends of these wires can be gently bent flush with the bone so as not to irritate nearby tendons, muscles, and nerves. The ends of the pins are cut leaving 1-2 cm of pins. A pin-bender is next used to bend the ends of the pins in a proximal direction, and the ends of the pins are cut again so that approximately 5-8 mm of each pin remains. This final bending maneuver prevents the tension band from slipping off of the pins during the post-op period. After correct pin and tension band application, a distraction load on the avulsed bone fragment tensions the figure of eight wire, which effectively resists the distraction force. When performing pin and tension band fixation, it is important to pins and wire of appropriate size. The most common causes of pin and tension band failure are small/improperly placed pins, small/improperly tightened wire, or placement of the transverse bone tunnel too close to the periosteum/cortex of the bone, resulting in wire pull through during the recovery period.

When performing pin and tension band fixation on the tibia tuberosity, it is important to begin both pins at or above the proximal aspect of the tibia tuberosity, and aim pins toward the caudal and medial aspect of the tibia. This ensures maximal bone purchase and reduces the incidence of pin-induced fracture of the tibia tuberosity.

Contraindications of Cerclage

Cerclage wires are contraindicated in the treatment of transverse, short oblique, segmental or comminuted fractures. When evaluating pre-operative radiographs of fractures, all of the fragments, even tiny ones, should be counted and if there are more than two fragments, another method of fixation should ideally be considered. Again, the exception is a three-piece fracture in which the third fragment is a large butterfly fragment. Cerclage wires are also contraindicated if, for any reason, the full 360 shaft cannot be re-constructed, or the shape of the bone is such that they cannot be applied so that they will remain perpendicular to the long axis of the bone without slipping. Loose or damaged cerclage wires should always be removed. Finally, the surgeon must consider the risk of additional damage to the blood supply when considering cerclage wire in high-velocity, open or infected fractures. IM pin and cerclage wire fixation is contraindicated for these types of cases.

Complications and their Prevention

Properly applied cerclage wires rarely cause problems, however, *improperly applied wires almost always cause problems*. The most common complication of cerclage wiring is the development of loose wires during recovery. Loose wires occur either as a result of failure to completely reduce the fracture, improper tightening techniques, or entrapment and subsequent necrosis of soft tissues. In the author's experience, cerclage wire complications and failures most commonly occur when utilized inappropriately on short-oblique or multi-fragmented fractures. The most common scenario is the selection of IM pin and cerclage wire stabilization for transverse, short oblique, or comminuted fractures in medium to large breed dogs. Lastly, loose wires prevent revascularization of the fracture, and sequestration of non-viable bone fragments often results. This event dramatically increases the risk for post-operative osteomyelitis, which results in lameness, swelling, and incisional draining or development of draining tracts after surgery.

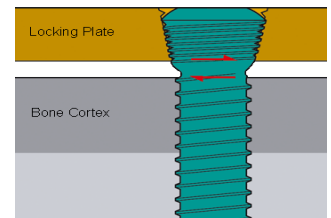
Conclusion

Prevention of complications with IM pins and cerclage wire requires excellent pre-operative fracture decision making, meticulous surgical technique, and appropriate post-operative exercise restriction for the patient. A failed pin and cerclage wire fixation can be devastating for the patient - in some cases irreparable, even if referred to a specialist with access to a variety of implants and techniques. Practice using plastic bones, broom handles, and cadaver limbs is strongly suggested for the inexperienced surgeon in order to gain experience and confidence with this technique.

Bone plates have been used effectively for treatment of fractures in dogs and cats for over 30 years. Over the years, different designs of plates have become available, including DCP (dynamic compression plates), LC-DCP (limited contact dynamic compression plates) and special situation plates (veterinary cuttable plate, acetabular plates). The most recent plate design to become available is the locking compression plate. The unique feature of the locking plate is the presence of a threaded plate

hole that couples with a threaded screw head or alternate locking mechanism. A number of companies have developed locking plates. One of the most recognizable locking plates is the Synthes LCP.

The Synthes LCP features a combi hole; this is a plate hole through which the surgeon can apply compression using a standard cortical screw or apply a locking screw. Using a locking screw, threads in the head of the screw engage threads in the plate hole, locking the screw to the plate. The ability to lock the screw to the plate increases the stiffness of the construct and the pull out strength of the bone plate and screws. Standard plates do not have threaded holes; stability is achieved through compression applied between the plate and bone surface when tightening the screws. The friction between the plate and the bone provides the stability to the bone-implant construct. In contrast, the locking plate achieves stability through the concept of a fixed-angle construct. The locking plate is not pressed firmly against



Locking screws have a threaded head that locks the screw to the locking plate.



Locking plates have a dual purpose hole that can accommodate a locking or cortical screw.

the bone as the screws are tightened. The locking screws and plate function more like an external fixator. The plate functions as a connecting bar and the screw functions as a threaded fixator pin. The threads in the head of the locking screw engage the hole of the plate, similar to the clamp of an external fixator.

EQUIPMENT AND APPLICATION

Many of the same instruments used for application of traditional plates are used for locking plates. The 3.5 mm locking plate and screws are most commonly used in small animals. The 3.5 mm locking plate will accommodate 3.5 mm cortical screw, 4.0 mm cancellous screws or 3.5 mm locking screws. A 2.8 mm drill bit is used when applying 3.5 mm locking screws. A guide is screwed into the intended hole to center the hole when drilling. The locking screw is self-tapping and is placed with a hand or power driver.

TraumaVet system is a locking system for internal fixation. It consists of a steel support, with threaded holes where threaded inserts can be secured. The inserts are externally threaded and are to be screwed into the support. The inner hole of the insert is conical to lodge the head of the screw. The support together with the inserts forms the plate. The screw is a self-tapping with angular stability. The screw head is conical, coupling with the insert to achieve the locking mechanism.



The unique conical screw-plate locking system ensures the distribution of force throughout the structure, virtually eliminating the risk of implant breakage and screws backing out.

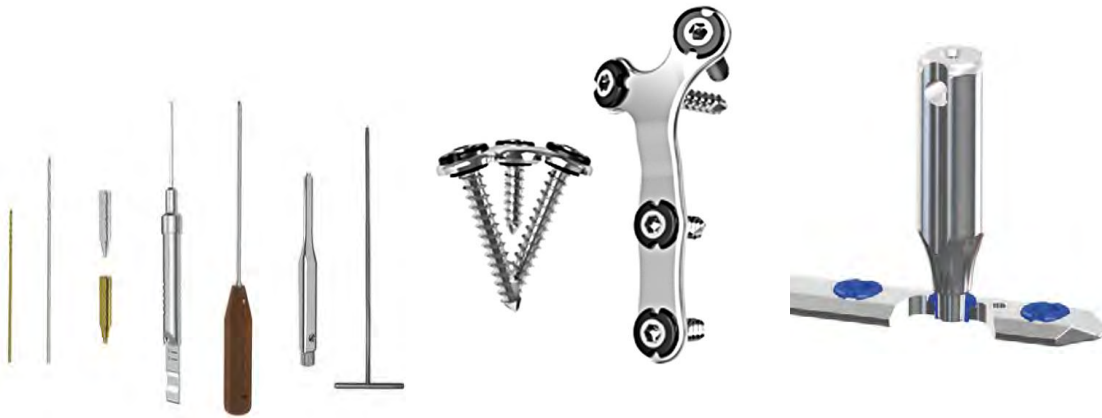
The locking system allows easy locking of the screw in the plate and eliminates the possibility of cross-threading the screw head in the plate.

The plate thickness ranges from 1.2mm to 2.0mm in the mini series and from 1.5mm to 3.0mm in the medium series.

The Fixin bone plate system is ideal for minimally-invasive fracture repair, direct fracture reduction, indirect fracture reduction and corrective osteotomy to treat angular limb deformities.

Traditional locking plates use a threaded screw-plate locking system. TraumaVet screws have a 2° conical head which locks into a corresponding 2° tapered cone in the bushings, which are pre-assembled on the plate. This coupling eliminates the risk of cross threading between the screw and plate and ensures a simple operative technique. It also permits

easy removal of the screws. It is an extremely stable coupling, which effectively transfers loads through the structure and reduces the risk of screws backing out or plate breakage.



INDICATIONS for application of Locking plates

Locking plates are particularly useful when screw pull-out is at a greater risk. Screws may be susceptible to pull-out failure in the metaphyseal region of bones where the bone cortex is thin, in osteoporotic bone, older patients, patients having slow bone healing conditions and patients that have poor compliance to restricted activity during the postoperative period. A locking plate is also useful with the presence of a limited proximal or distal target (epiphyseal or metaphyseal fracture). Minimally invasive plate osteosynthesis (MIPO) is best accomplished with the application of a sliding plate technique. MIPO is usually accomplished with small proximal and distal portals where one applies two – three screws in each fracture segment.

Minimally invasive technique is an open technique whereby direct or indirect fracture reduction is achieved with small exposure portals. Each portal is strategically located to allow proper reduction and application of an implant. An incision (1-2cm) is made overlying the metaphyseal-epiphyseal area of the proximal and distal parent bone. Soft tissue is reflected to expose the bone surface where the implant will be applied. An additional small portal may be necessary to expose the transverse (short oblique) fracture site. A periosteal elevator is used to create an avenue on the surface of the bone for positioning the implant. The fracture is visually aligned and the implant applied. With bone plates and screws, the implant is slid beneath the soft tissue into the previously created avenue on the surface of the bone. One proximal and distal screw is applied to hold fixation while alignment is examined. The remainder of screws is then inserted. As a generally rule, 2-3 screws are inserted into each fragment.



The Tennessee Board of Veterinary Medical Examiners

Music City Veterinary Conference 2016

Helpful Resources

The website for the Board of Veterinary Medical Examiners:

<http://tn.gov/health/topic/vet-board>

The Rules of the Board:

<http://share.tn.gov/sos/rules/1730/1730.htm>

Policy Statements from the Board:

<http://tn.gov/health/article/vet-policies>

TVMA website:

<http://www.tvmanet.org/>

The Tennessee Veterinary Practice Act:

<http://www.lexisnexis.com/hottopics/tncode/>

Contact the Board of Veterinary Medical Examiners

615-532-5090 local or

1-800-778-4123 nationwide or

665 Mainstream Drive, 2nd Floor
Nashville, TN 37243

Article from dvm360 newsmagazine:

“What Gives Veterinary Boards the Right?”

<http://veterinarynews.dvm360.com/what-gives-veterinary-boards-right?rel=canonical>

My Top Ten Suggestions for Avoiding Complaints with the Tennessee Board of Veterinary Medical Examiners

No one wants to deal with complaints brought before the Board. They are stressful, time consuming, and can be costly, regardless of the outcome. While sometimes complaints are unavoidable, here are some things that can decrease your chances of having to deal with a Board action and can increase your chances of a successful outcome.

1. **Know your Practice Act.** The Practice Act contains the laws that govern the practice of veterinary medicine. It is important to stay current and stay in compliance.
2. **Know the Rules and Regulations of the Board.** If the Practice Act is the foundation of the house called veterinary medicine, the Rules are the bricks and mortar. Stay up to date on the rules and watch for any rule changes.
3. **Know the Policy Statements of the Board.** Following on with our analogy about the house, think of the policy statements as being the verbal directions given by the owners of the house. They serve as further clarification of a particular Board's stance on an issue and guide future Board decisions and actions.
4. **Communicate with your clients.** A breakdown in the lines of communication between the veterinarian and the client often leads to client dissatisfaction and can result in the filing of a complaint. Maintain those open lines of communication, interact with clients courteously and professionally, and make sure your staff follows your example.
5. **Educate and properly supervise your staff.** Ultimately, you are responsible for the things your staff does. Be sure that staff members are performing within the boundaries of the Act and the Rules.
6. **Keep good records.** Be sure that your documentation is not just adequate, but is excellent. Making notes of negative findings can be just as important as recording positive findings. Your recommendations that are declined by the owner should be recorded.
7. **Don't let problems fester.** The longer you wait to deal with a disgruntled client or to address a complaint with the Board, the more trouble it will be in the long run. When dealing with our clients and their pets' medical problems, we tell them all the time that it is better to get out ahead of a problem and not let a minor illness turn into a life threatening disease. The same applies here.
8. **Interact with your colleagues and promote professionalism.** Too often we lead very isolated and insular careers. Collegiality amongst your fellow

veterinarians, especially those in your immediate area, can be rewarding and it is much less likely that a friend will register a complaint with the Board than an enemy.

9. **Get legal advice.** If you are faced with a Board complaint that is not easily resolved, retain an attorney familiar with the Health Related Boards and the Uniform Administrative Procedures Act. Consider malpractice insurance that covers Board matters. This is sometimes referred to as “license defense coverage” and is in addition to your veterinary malpractice insurance.
10. **Practice to the standard of care.** The standard of care is defined as “The ordinary level of skill and care that any health care practitioner would be expected to observe in caring for patients.” Remember that the standard of care may also vary based on practice type, your level of advanced training, the region of the state or country, and other factors.

URINARY SURGERY TIPS AND TRICKS

Patricia A. Sura, MS DVM DACVS-SA

Basic Surgical Principles

No matter the surgical procedure performed on the urinary tract, there are certain principles that require adherence. It is important to achieve adequate visualization of the area of interest, while simultaneously preventing contamination of nearby tissues with urine. The approach to the surgery should allow for both correction of the issue at hand, as well as collecting appropriate samples for analysis. Closure of the urinary tract is best performed with fine, monofilament absorbable suture, and urinary diversion can be protective and promote healing.

Cystotomy

Perhaps the most familiar surgical procedure for removal of uroliths, open cystotomy is readily performed via a midline celiotomy. The advantages of surgical cystolith removal are that the results are rapid, a diagnosis of cystolith type is easily obtained, and risk of obstruction is removed.¹ However, cystotomy is an invasive procedure. Therefore, if medical or minimally invasive therapies are an option, they should be considered prior to intervention.

Following a midline approach to the abdomen, the urinary bladder is isolated and surrounded with moist laparotomy sponges to protect the viscera from inadvertent spillage of urine. Cystotomy is classified as a clean-contaminated procedure; therefore prophylactic antibiotics are indicated. Stay sutures are used to harness and manipulate the bladder. The safest and easiest approach into the urinary bladder is on the ventral surface, away from the ureteral openings. A stab incision is made into the lumen, and suction used to remove urine from the bladder. The incision is then lengthened carefully on midline with a blade or scissors. A cystotomy spoon can be used to gently manipulate stones out of the neck of the bladder and proximal urethra. In male dogs, the prepuce is flushed prior to surgery with 0.05% chlorhexidine diacetate.² A urinary catheter is passed during the procedure, and flushed multiple times to ensure that there are not stones trapped in the urethra. In female dogs, a large catheter can be placed antegrade to ensure complete urolith removal. Closure is with an inverting pattern or appositional pattern. A single layer of suture is as effective as a double-layer closure. 3-0 to 4-0 is generally sufficient for companion animals, and the smallest gauge required to maintain strength at the incision is desired. If infection is suspected, or if the patient has any disease processes that may delay healing of the urinary bladder, PDS or Maxon may be preferred to Monocryl, as Monocryl degrades quickly in infected urine.³

Following cystotomy for urolithiasis, post-operative radiographs are imperative, as up to 20% of companion animals may have residual stones after surgery.⁴ Post-operatively, fluid diuresis is important, to prevent obstructive blood clots from forming in the urinary bladder. Urge incontinence, hematuria and pollakiuria will be noted for several days after surgery, and should decrease precipitously with time. Complications include recurrent obstruction (stones, blood clots) and uroabdomen.⁵ In a recent study of 144 cases of open cystotomy, the incidence of uroabdomen was 1.4%.⁶ Urothelium begins to migrate across incisions in 24 hours, and is complete by 96 hours. 100% of original strength is acquired within 14-21 days.

Cystoscopic-Assisted Cystotomy

Minimally invasive options are becoming increasingly available in the veterinary field. This is perhaps most notable in the area of urinary surgery. Due to the nature of the urinary tract, endoscopic examination is relatively easy. Over the past five years or so, urethroscopy and cystoscopy have transitioned from exclusively diagnostic tools to therapeutic tools with success rates that approach or exceed traditional surgical methods.⁶

Traditional surgical methods result in incomplete stone removal up to 20% of the time,⁷ the magnification and complete view afforded by cystoscopy should reduce this percentage, especially in the face of radiolucent stones. Similarly, suture nidus cystoliths represent up to 9.4% and 4% of recurrent cystoliths in dogs and cats respectively.⁸ The minimal incision will therefore be beneficial to many patients.

Cystoscopic-assisted cystotomy is performed through a 1-2 cm midline incision, made at the level of the apex of the palpable urinary bladder. Rather than use a laparoscope to assist in urinary bladder identification and entry, which requires additional specialized equipment and establishment of pneumoperitoneum, cystoscopic-assisted cystotomy relies on the surgeon to isolate the apex of the urinary bladder and temporarily fasten it to the linea alba, where cystoscopy and stone removal can be performed through a small stab incision.

A recent report of the use of this technique in 27 cases indicated a procedure time of 50-80 minutes. One patient had a residual urolith on radiographs that had to be retrieved with a second procedure. No major short- or long-term complications were reported.⁹

Urinary Bladder Biopsy:

Biopsy of the urinary bladder is simple to perform, and can be extremely useful not only for histopathologic analysis, but also for bacteriologic culture and sensitivity. Mucosal biopsy has been shown to be positive in dogs with bacterial urinary tract infection and urolithiasis more frequently than cystocentesis (14 of 16 cases versus 12 of 16 cases).¹⁰

Cystostomy Tube Placement/Management

Since urine diversion favors wound healing, cystostomy tubes can be used either for pre-operative stabilization, post-operative diversion, or for management of chronic urethral obstructive disease.¹¹⁻¹³ When used in a short-term manner, the stoma is left to heal via second intention. Following placement in 10 dogs and one cat, micturition returned to normal after tube removal, with leakage from the stoma for up to four days while granulation occurred.¹²

Common complications of tube cystostomy, especially when used for long-term management of urethral obstruction include urinary tract infection, inadvertent tube removal and stomal infection or inflammation.^{12,13} Low profile tubes can be substituted for traditional cystostomy tubes to minimize mutilation of the tubes by the patient. However, an e-collar should be used whenever

the patient is unsupervised. Should the patient inadvertently remove the tube, time is of the essence for replacement, since the stoma tends to contract rather quickly.

Nephrectomy

Removal of a kidney can be a salvage procedure for nephroureterolithiasis, intractable pyelonephritis, renal hematuria, and renal neoplasia. It is important to discuss potential complications with the owner prior to surgery, including the potential for symptomatic renal failure if the contralateral kidney is diseased. Glomerular filtration rate is an excellent means of determining the safety of nephrectomy. Excretory urography may also be employed, however this test is qualitative not quantitative – it will show whether or not both kidneys are functional, but will not yield percent function.

To obtain adequate visualization of the kidney, pack the abdominal viscera behind the physiologic retractors – the descending duodenum on the right and the mesocolon on the left. It is important to identify both ureters, as they run in close association with each other, to ensure that the contralateral ureter is preserved. The kidney is then released from its peritoneal attachments, and rolled medially to expose the hilus. The renal artery is double ligated, as is the renal vein. In intact animals, the left gonadal vein is a tributary of the renal vein and should be preserved. The ureter is then ligated and transected at its junction with the urinary bladder and the kidney is removed.

Ureteral Bypass

This surgically placed device consists of a locking-loop pigtail catheter placed in the renal pelvis and tunneled subcutaneously to a reservoir button secured to the lateral abdominal wall. A second catheter then travels from the reservoir and is passed through the abdominal wall and inserted into the apex of the urinary bladder. Use of this device essentially bypasses the entire ureter. These devices are easy to place, but data regarding their use is lacking in the veterinary literature.¹⁴

Primary Urethral Repair

Anatomically, the proximal urethra is contained within the abdominal cavity, and compromise can lead to uroabdomen. Damage to the intrapelvic and distal urethra can cause urine extravasation under the skin with subsequent slough. This usually presents as bruising, swelling, erythema and pain progressing dependently down the hind limbs.

The urethra has an amazing capacity for healing. Therefore, conservative management of urethral tears is possible. Urethral healing relies on mucosal continuity, and lack of significant urine extravasation.^{15,16} A small, persistent strip of mucosa is all that is necessary to allow urethral healing, generally without stricture formation.¹⁵⁻¹⁷

Primary alignment has been described in 10 cats, in which a urinary catheter was used to span the ruptured area for 5-14 days. A 100% success rate for urethral healing was reported, with a single case of urethral stricture.¹⁸ An appropriate-sized catheter should approximate the diameter of the urethra without exerting outward pressure, as this may slow wound healing. If a

retrograde catheter cannot be passed, a laparotomy may be useful to pass a urinary catheter from inside the bladder through the urethra. A second urinary catheter can then be attached to the end of the first and pulled into the bladder.^{18,19}

Should leakage persist following conservative management, or if primary repair is chosen, fine gauge (4-0 or smaller) suture material is used to repair the defect. Pubic osteotomy or ostectomy may be required to access the intrapelvic urethra.^{17,20} Urethral resection and anastomosis is necessary in cases of complete transection of the urethra. Interrupted sutures can be pre-placed, to facilitate inclusion of the mucosa and submucosa. A urethral catheter is left in place for 3-5 days to divert urine away from the operative site. Stricture and dehiscence are the most common complications.

Prescrotal Urethrotomy

This technique is used to remove stones at the base of the os penis. As most stones can be hydropulsed into the urinary bladder, this should be attempted first. General anesthesia is necessary for adequate hydropulsion, as it affords maximal urethral relaxation. For stones that cannot be hydropulsed, a small incision is made at the base of the os penis, directly over the stone. The retractor penis muscle is elevated, and the urethra is incised on midline. If the urethra appears to be healthy and minimally traumatized, the incision is closed with 4-0 or smaller interrupted absorbable sutures. If severe trauma to the urethra is present, the incision can be left open to heal by second intention.^{21,22} If the stone cannot be removed, or if stricture occurs, a scrotal urethrostomy will be required.

Scrotal Urethrostomy

Urethrostomy is the permanent creation of a new urethral orifice in dogs to bypass stricture formation (usually at the base of the os penis), recurrent obstruction with urethroliths, or in cases of penile trauma. Creation of a new large stoma will hopefully also allow future uroliths to pass. The scrotal location of the urethrostomy is chosen as the urethra is very superficial in this location, with much less cavernous tissue. In addition, this placement allows the stoma to be directed ventrally.^{21,23} Principles of urethrostomy must be adhered to in order to achieve best results. These include:

- Tension-free closure
- Incision of the urethra on midline to ensure appropriate amount of tissue for closure
- Meticulous apposition of urethral mucosa to the skin
- Creation of a large urethrostomy opening to allow diminished size expected after healing
- Prevention of self-trauma

The patient is placed in dorsal recumbency. An elliptical skin incision is made in the scrotal area, using care to ensure that there is sufficient tissue for closure without tension. If necessary, the patient is neutered. Using blunt dissection, the paired retractor penis muscles are localized and lateralized. If possible, a retrograde urethral catheter is placed to facilitate incision of the urethra on midline. A #11 or #15 blade is used to start the incision, which is then extended using iris scissors (or similar). The stoma should be 5-8 times the width of the urethra (usually a total

of 2-4 centimeters). During healing, the stoma is expected to diminish in length by $\frac{1}{3}$ to $\frac{1}{2}$. The urethral mucosa is then sutured to the skin.^{21,23}

Simple continuous suture patterns have been recommended, both to decrease incisional bleeding as well as subcutaneous bruising.²⁴ A bite of the fibrous tunica albuginea can also limit hemorrhage and provide strength to the closure. Continuous closure has been shown to decrease active bleeding from 4.2 to 0.2 days.²⁴ Bleeding during urination is still expected for several days after the procedure. Appropriate sized monofilament suture (4-0 or smaller) is used for closure; both absorbable and non-absorbable have been described.

It is important to outfit the animal with an Elizabethan collar prior to complete anesthetic recovery. Self-mutilation is extremely common, and is catastrophic in these cases with respect to continued bleeding as well as scarring and stricture of the orifice. Similarly, staff that will be working with the animal should be advised that the clot at the surgical site not be disturbed. Other complications include an increased incidence of urinary tract infection, likely from the reduced length of the urethra, and re-obstruction with calculi. A study evaluating long term outcome of scrotal urethrostomies in dogs found a 15% infection rate.²⁵ Treatment should be directed via culture and sensitivity results.

Perineal Urethrostomy

This is the procedure of choice to treat recurrent urethral obstruction in male cats, obstruction that cannot be relieved with catheterization, and distal urethral tears created iatrogenically during attempts to unblock cats.

An elliptical incision is made around the scrotum and penile body. The penis is bluntly dissected to the level of the bulbourethral glands. It is essential that the ischiocavernosus muscles and ventral penile ligament are completely transected to free the penile body from the pelvis. A gloved finger should easily pass from the 3:00 position ventrally and across to the 9:00 position without encountering any resistance. Minimal dissection is carried out dorsally, to prevent damage to the nervous structures which could lead to potential incontinence. The urethra is incised on midline with sharp iris scissors to the level of the bulbourethral glands, and far enough forward that a pair of curved hemostats could be passed into the urethra to the level of the box locks. The mucosa is again sutured to the skin with fine monofilament suture (5-0 to 7-0) in a continuous pattern. The dorsal sutures are the most difficult to place, and are often pre-placed in an interrupted fashion, followed by continuous closure of the rest. The distal penis is amputated, and the sutures are allowed to dissolve over time. Therefore, a rapidly dissolving suture material is preferred. If there is any concern about tension on the operative site, especially in an obese cat, a urethral catheter can be placed to avoid subcutaneous urine leakage. A large clot will appear over the incision which should not be disturbed, and the cat should wear an e-collar at all times for 2-3 weeks.

Infection rates following permanent urethrostomy can approach 53%.²⁶ In a study of 59 cats that had undergone perineal urethrostomy, there was a 25% complication rate within the first month of surgery, including stricture formation, urinary tract infection and perineal urine leakage.²⁷ In general, complications from urethrostomy include stricture formation, urine leakage into the

subcutaneous space, chronic urinary tract infection, perineal hernia and possible incontinence.^{27,28} In cats with perineal urethrostomy, complete dissection to the level of the bulbourethral glands will prevent stricture formation. In a study of failed urethrostomies, 8/11 cats with stricture formation had evidence of incomplete dissection and improper surgical technique.²⁸ Other causes of stricture formation are inappropriate apposition of the mucosa to the skin resulting in scarring, and self-mutilation by the patient.^{25,28}

Urethral Prolapse Repair

Urethral prolapse is an uncommon condition in intact male bulldogs and other brachycephalics. While the exact cause is unknown, proposed etiologies include urethritis, cystitis, other inflammation or excess sexual activity.^{21,29} The prolapse is seen at the tip of the penis, and is essentially the mucosa turned inside-out. It appears as a reddish to purple, cauliflower like lesion. Hemorrhage from these prolapsed areas is common.

Treatment of this condition is surgical, and includes neutering to decrease sexual drive, as well as either prolapse resection and anastomosis, urethropexy, or penile amputation and scrotal urethrostomy. A urine culture should be submitted, as infection can exacerbate this condition. Recurrence is problematic, especially if the animal is not castrated at the time of surgery, or if the dog was not appropriately managed after surgery. As with all urethral surgery, hemorrhage is common and stricture can be seen. Management is similar to dogs with scrotal urethrostomies (see above).

To resect the prolapse, the prepuce should be retracted to expose the penis. The dog is catheterized with a red rubber catheter to facilitate resection. Fine scissors are used to remove the prolapse. A cut– and –sew technique is used to appose the mucosa to the skin in 1/3 circumference intervals. A simple continuous pattern of fine absorbable suture is used.³⁰

Urethropexy has also been described, where the prolapse is reduced, and a suture is passed through the penile body and urethra to maintain reduction.^{29,30} Post-operative care is similar to that of other urethral surgeries. If an underlying cause of the prolapse is determined, appropriate therapy is necessary to prevent recurrence.

Urethral Occluders

Silicone implants are available that consist of an inflatable cuff that can be placed around the pelvic urethra and attached to a reservoir that is tacked to the lateral abdominal wall. Suitable for urethral sphincter mechanism incontinence (spay incontinence, residual incontinence after ectopic ureter repair, etc...), these devices are placed via a midline abdominal approach. The urethra is measured, and an appropriate device placed once the urethra is bluntly dissected from the surrounding adipose tissue using right angle forceps. The filling volume of the occluder is recorded, and the occluder secured in place without inflation of the device. Continence is improved in many dogs during the first month after surgery. After that time, if incontinence is still noted, small volumes (0.1 to 0.2 mL) of sterile saline are infused via a Huber needle into the subcutaneous reservoir, thereby filling the cuff. Initial results in four dogs have been published, with 3 of 4 completely continent at two years.³¹

Recessed Vulva

A recessed, or juvenile vulva has also been associated with urinary tract infections. This anomaly is seen primarily in medium- to large-breed dogs in which ovariohysterectomy was performed before one year of age.^{32,33} In one study, 12/22 dogs with recessed vulva had culture-positive urinary tract infections.³² This conformational deformity is thought to contribute to lower urinary tract clinical signs by causing urine retention within the vagina, and irritation of skin folds resulting in perivulvar dermatitis, incontinence and infection.^{32,33}

Vulvoplasty is a reconstructive procedure by which a crescent-shaped piece of skin and associated subcutaneous tissue is removed between the anus and vulva. Resultant elevation of perivulvar skin and closure of the wound acts to evert the vulvar lips and expose the vestibular opening. This procedure was successful in resolving urinary tract infections in 84% (16/19) of dogs.³³

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GASTROINTESTINAL SURGERY TIPS AND TRICKS

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Principles

When considering gastrointestinal surgery, contamination is of prime concern. Every effort should be made to minimize exposure of intestinal contents to the abdominal cavity. All clean procedures (hepatic biopsy, lymph node removal, etc...) should be performed prior to techniques that enter the GI tract. Proper isolation of the segment of tract to be opened is crucial. Stay sutures and atraumatic forceps should be used for manipulation. Abrasion and dessication can increase adhesion formation and reduce GI motility. Moistened laparotomy pads should be used as a barrier to cordon off the GI segment in question from the rest of the abdomen. This will also prevent dessication of the tissues. A set of clean instruments should be set aside prior to entry of the GI tract, and used for abdominal closure. Gloves should be changed prior to closure of the abdomen. Powder should be rinsed from the outside of gloves to reduce granuloma and adhesion formation. Frequent and copious lavage is essential. Use warm fluid. Fluid that is too hot may increase adhesion formation and cause vasodilation with subsequent hypotension. Cold fluids exacerbate hypothermia. Evacuate all fluid prior to closure. Residual fluid inhibits immune clearance.

Should a breach in technique occur, it is beneficial to have chosen appropriate antibiotics for the segment of GI tract entered. Antibiotics should be given at the time of anesthetic induction, and dosing may be repeated during lengthy procedures. For gastric procedures, first generation cephalosporins (e.g. cefazolin) or aminopenicillins are adequate, due to low pH and minimal bacterial inhabitants. As transit through the small intestine occurs, gram positive and gram negative inhabitants occur in increasing numbers. Therefore, appropriate spectrum, such as ampicillin/enrofloxacin, or higher generation cephalosporins are indicated. Particular attention is paid to anaerobes once the ileum/large intestine is affected.

Suture choice recommended for the majority of GI surgery is 2-0 to 4-0 absorbable monofilament. Swaged needles result in the smallest holes through tissue, and taper needles provide the most protection against tissue tearing. It is important to ensure that the submucosa is engaged, as this is the holding layer of the gastrointestinal tract. Full-thickness, appositional suture patterns are placed with the most ease, and are not disadvantageous to inverting patterns. Non-absorbable suture may be used if delayed wound healing is anticipated, but has been associated with entrapment of foreign bodies with subsequent obstruction in rare cases. Braided suture may encourage bacterial colonization, and serve as a nidus. Braided suture also causes increased inflammation when compared to monofilament. This may prolong the lag phase and delay wound healing. Continuous suture patterns provide adequate strength when applied properly, and leave less foreign material in the wound. Various stapling devices are also available for GI surgery.

Preoperatively, it is important to correct fluid imbalances. Crystalloids can be used for immediate volume; however approximately 75-85% moves into the interstitial space within one hour of administration. Combination use with colloids will allow fluid to remain in the intravascular space, as well as attract fluid from the interstitium. Electrolyte imbalances are corrected, and additional pharmacologics can be administered, based on the level of the

gastrointestinal tract affected. Last, but certainly not least, the majority of gastrointestinal maladies are incredibly painful, so appropriate analgesic therapy is vital.

The “danger zone” of gastrointestinal healing is between days 3-5 for gastric and small intestinal incisions, and can be as long as seven days with large intestinal procedures. This is due to the lag phase of healing, generally accepted as post-operative days 0-4, in which holding strength is provided by suture. The proliferative phase of gastrointestinal wound healing occurs between days 3-14. During this time, there is a rapid increase in wound strength, which approximates normal in 10-17 days. Finally, collagen remodeling occurs during the maturation phase, from post-operative day 10-120.

Biopsy technique

The ideal technique for gastrointestinal biopsy includes obtaining a full-thickness sample of adequate size in such a manner that artifact is minimized. In addition, there should be minimal complications associated with the biopsy technique. There are two techniques that have yielded excellent histopathologic results in my hands, with minimal complication. The first involves use of a sharp (preferably new) 4-6 mm biopsy punch. Care must be used to avoid damage to the mesenteric surface from overzealous application. This technique is most useful in areas where the intestinal wall is extremely thick, as it allows controlled sampling of all layers without fear of missing the mucosal layer, or creating a very large defect.

Another technique that is simple, and very effective in more normal pieces of gastrointestinal tract involves the use of a stay suture for manipulation, which minimizes crush artifact. It also allows the wedge biopsy to be removed with a scalpel blade, further minimizing artifact when compared to use of scissors. Due to the tapering nature of a wedge biopsy, a common error is to include too small of a piece of mucosa; ensure that the angle of removal is not too steep.

Gastrointestinal biopsies are closed in a simple interrupted or continuous manner with appropriate suture material. Mucosal eversion can be trimmed, or a modified Gambee pattern used to invert the mucosa while apposing submucosa. However, this is not essential, as there is no difference between an appositional and inverting pattern. The most important aspect of any suture pattern is incorporation of the submucosa in the closure. Complications of intestinal biopsy include leakage, dehiscence and possible stricture. If a grossly abnormal area is to be biopsied, consider excisional biopsy (resection and anastomosis) with apposition of healthy tissue, rather than incisional biopsy, as wound healing capacity may be diminished.

Patching

Any GI repair can be fortified with either an omental or serosal patch. Omentum brings blood supply, angiogenic factors, immunocytes and physiologic drainage to an incision. The simple act of tacking a portion of omentum to a wound may increase the likelihood of a good seal over allowing physiologic migration. Do not tack omentum circumferentially around a loop of intestine, as strangulation may occur. Serosal patching is accomplished by gently laying loops of intestine over a defect, and suturing them in place.

GDV/Gastropexy

Gastropexy is the treatment of choice for gastric dilatation, with or without volvulus. In this condition, accumulation of gas in the stomach occurs primarily from aerophagia. Fermentation leads to further distension, and inflow/outflow obstruction is certainly a component. It is unclear as to whether volvulus is secondary to gas accumulation, or whether it occurs early in the disease process, causing obstruction and gas accumulation.

Breeds at increased risk for this condition are Great Dane, Irish Wolfhound, German Shepherd, Rottweiler, Doberman Pinscher, Newfoundland, Irish Setter, Akita, Weimaraner, Saint Bernard and Standard Poodle. It has been shown that an increasing thoracic depth:width ratio may predispose animals to the disorder. A number of risk factors have been suggested, including genetics, thoracic depth:width ratio, food particle size, number of meals per day, exercise after meals, prior splenectomy, attitude, etc...

GDV is definitely a surgical disease. In one study, 66% (55/83) of animals survived initial decompression, but without surgery 71% experienced recurrence, and of this 71%, 77% died or were euthanized due to the disease process. A second study reported a 4.3% recurrence of GDV when gastropexy was performed, vs. a 54.5% recurrence rate with conservative management.

There are many techniques described for gastropexy. The technique used should lead to a permanent adhesion, with minimal complications. The appropriate area for pexy is just caudal to the 13th rib, approximately 8 cm to the right of midline. AVOID incorporational gastropexies unless the patient is absolutely dying before your eyes! In this technique, the stomach is incorporated into the linea alba closure. This almost guarantees entry and contamination of the abdomen if celiotomy is again warranted.

Gastrotomy

Gastrotomy is typically performed for foreign body removal. The incision should be made in the fundus, away from the major branches of the gastroepiploic vessels. Intraluminal hemorrhage is quite common with this technique, and may be minimized with a two layer closure – the first involving the mucosa and submucosa, and the second involving the muscularis and serosal layers.

Resection and anastomosis

This procedure is most often performed for foreign body removal, but is also employed for neoplasia, intussusception, and stricture removal. The goal is to re-appose healthy tissue. The most common complication is leakage and dehiscence, which usually occurs on the mesenteric border, due to obscured visualization of suture placement by fat. Tips to reduce this complication include placement of a mesenteric and antimesenteric stay suture first, and using these to manipulate the segments of bowel, leak testing anastomotic sites, and approximating luminal diameter. Means of luminal approximation include spatulating the smaller bowel loop, oversewing the larger bowel loop, and cutting the smaller bowel loop on a steep angle to approximate the larger lumen. If the loop is cut on an angle, be sure that the mesenteric base is the longer side of the angle, to provide maximal blood supply. Finally, when luminal disparity is mild, suture placement can be used to minimize differences.

Treating peritonitis and hypoproteinemia appropriately is crucial for anastomotic healing, as well as prevention of dehiscence. A study of 121 dogs revealed a higher incidence of leakage when resection and anastomosis or enterotomy was used to remove foreign bodies, and when hypoproteinemia was present. This was confirmed in a study of 115 dogs and cats. Pre-existing peritonitis was also found to be significantly associated with dehiscence. Finally, with any gastrointestinal surgery, early enteral nutrition is associated with healing, as enterocytes receive much of their nutrition from

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BASICS OF WOUND MANAGEMENT

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Under ideal circumstances, wounds heal in a predictable manner. It is essential to understand the process of normal wound healing, so that wounds are managed appropriately, and complications are recognized. Wound healing can be divided into four overlapping categories: *initiation*, *inflammation/debridement*, *proliferation* and *maturation*. It is important to recognize that wound healing is a fluid process, and the phases described below occur on a continuum until the wound has completely healed. Rather than thinking of these phases having a distinct beginning and end, consider instead that they occur in peaks and valleys, with some stages predominating at any given time, but not in absence of the others.

In a nutshell: The initiation phase describes the local response to wounding. Paracrine factors (substances secreted by one cell type that attract other cell types) and autocrine factors (substances from one cell type that attracts like cells) then result in the arrival of inflammatory and proliferative cells to the wound. Dead tissue and contaminants are removed, while fibroblasts begin the repair process. Capillaries are restored, collagen is produced, and the wound begins to contract. Finally, epithelial cells migrate over the wound surface, restoring the environmental barrier. During maturation, collagen remodeling and rearrangement results in a significant increase in wound strength.

Initiation: As the name implies, this stage begins immediately at the time of wounding. Bleeding serves to cleanse and moisten the wound surface. In response, the body immediately secretes vasoconstrictive compounds to minimize blood loss. This constriction is short-lived, lasting only minutes. The vessels then begin to dilate, allowing platelets to congregate with blood and fluid and create a clot. The clot serves to arrest bleeding, as well as provide a template on which further wound healing can occur. While the clot provides a mechanical resistance to infection, it does not contribute to wound strength. The surface of the clot dries into a scab, below which the healing process continues. The dilated blood vessels contribute to cardinal signs of inflammation: heat, redness, and swelling.

Inflammation/debridement: This phase is characterized by the arrival of white blood cells into the wound defect. It typically begins within 6 hours of injury. Platelets that have arrived at the area of tissue disruption degranulate, releasing a host of cytokines. These cytokines are chemoattractants for various other cell types. In essence, the inflammatory cells serve to tidy-up the wound environment, and set the stage for healing. Neutrophils are the first leukocyte population to arrive, and can be measured in the wound bed at 24-48 hours. Platelet-derived growth factor (PDGF) is a potent chemotactic factor for neutrophils, as are fibrinopeptides, created from the conversion of fibrinogen to fibrin. Neutrophils are then activated by transforming growth factor beta (TGF- β), tumor necrosis factor alpha (TNF- α), interleukin one (IL-1), complement, and bacterial products. Neutrophils release potent reactive oxygen species, and are efficient bacterial killers. Superoxide and hydrogen peroxide are generated, which require a high partial pressure of oxygen at the wound surface. As the environment becomes oxygen-depleted, bacterial killing by neutrophils is impaired. Along with reactive oxygen

species, neutrophils also secrete proteases. These compounds serve to break down necrotic tissue, which is then phagocytosed along with wound bacteria. The proteases attract even *more* neutrophils to the wound. The combination of neutrophils, wound fluid and debris is commonly recognized as pus. Neutrophils, however, are not essential for wound healing. Monocytes migrate into the wound along with neutrophils and transform into macrophages. Additional monocytes are attracted via the oxidative burst and cytokine release from neutrophils. As neutrophils are short-lived cells, the population of white blood cells within the wound bed shifts over time, until macrophages predominate (46-72 hours after wounding). Macrophages are essential for wound healing. Macrophages produce important growth factors, including fibroblast growth factor (FGF), epidermal growth factor (EGF), platelet-derived growth factor (PDGF), TGF- α , TGF- β , tumor necrosis factor (TNF), and various interleukins. Monocytes can differentiate into epithelial cells and histiocytes, and are capable of proliferating within the wound. Macrophages also release matrix metalloproteinases (MMPs) that serve to degrade the extracellular matrix. Macrophages are efficient phagocytic cells, and continue to remove bacteria and wound debris.

Proliferation: Following the destructive nature of inflammation, tissue is replaced during the proliferative phase, which usually lasts about two weeks after wounding. The goal of the proliferative phase is coverage of the wound via replacement of soft tissue. This occurs via angiogenesis, fibroplasia and epithelialization. Vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), and other factors from the wound bed encourage vascular outcropping from surrounding vessels. Newly sprouted capillaries within the wound bed then serve to deliver oxygen and nutrients to the healing process. It is believed that this capillary network is also stimulated by low oxygen tension within the wound. Fibroblasts migrate into the wound under the influence of PDGF, TGF- β and EGF provided by the platelets and macrophages. These fibroblasts begin to synthesize type III collagen. Type III collagen is the weakest of collagen, but does add incredible strength to the wound bed. Type III collagen, fibronectin and hyaluronic acid within the wound bed is known as *ground substance*. Along with capillaries, fibroblasts and macrophages, a robust, vascular tissue known as *granulation tissue* is born. Granulation tissue consists of approximately 30% type III collagen. Granulation tissue is present within the wound at 3-5 days. TGF- β is responsible for differentiation of wound fibroblasts into myofibroblasts. These cells express actin, and are capable of contraction. The wound fibroblasts also transition into production of type I collagen, creating a durable connective tissue. Once the collagen content stabilizes, the fibroblasts undergo apoptosis, leaving a scar. Epithelialization begins at the margin of the wound, and appears as a light pink rim that gradually progresses inward. This can be seen beginning 4-5 days post-wounding. However, this is markedly affected by wound size, as sharply created incisions that have been sutured can be epithelialized within 1-2 days. Epithelial cells creep across the wound surface until they contact other epithelial cells. At this point migration ceases, and stratification begins. This phenomenon is known as *contact inhibition*. During the proliferative stage of wound healing, contraction is also occurring, which substantially reduces wound size. Contraction is dependent on the tension of the wound and surrounding tissue. As healing progresses and myofibroblasts decrease in the wound, contraction slows. The collagen that has been deposited does not have contractile properties. Contraction will also stop once the tension on the wound exceeds the capabilities of myofibroblasts.

Maturation: Wound maturation consists of remodeling collagen into a tough scar. There is essentially no appreciable wound strength during the first 3-5 days after injury. Historically, this was termed the “lag phase” of wound healing, although now we know that there is a LOT going on that sets the stage for proliferation and maturation. At the end of the proliferative phase, wounds achieve approximately 20% of their final strength. Proliferation was initially termed the “log phase” of healing, representing logarithmic acquisition of strength (see graph above). Maturation of the wound consists of massive collagen remodeling. The bundles become thicker and orient themselves along the lines of tension. This reorganization can take months to years. At maximum strength, a scar is 70-80% as strong as normal, unwounded tissue

The description of wound healing above is also known as “second intention healing” i.e. allowing the wound to heal on its own, via the process of contraction and epithelialization. When a wound is created surgically and then sutured closed, “first intention healing” occurs. By definition, a surgical incision is sharp, and causes minimal tissue trauma – crushing or devitalizing tissue in the process is not anticipated. Surgical wounds are also created under sterile conditions. There is minimal inflammation, and the wound size is markedly reduced through the application of suture material. The gap, or wound size, is one millimeter or less. Therefore, there is little need for fibroplasia and angiogenesis, epithelialization can occur rapidly, and there is minimal scarring. “Third intention” healing is a happy medium of first and second intention. This refers to suturing a wound once healthy granulation tissue appears. Clinically, wounds should progress in an orderly fashion. Purulent debris is expected early, followed by the appearance of granulation tissue at 3-5 days. As granulation tissue matures, it should appear robust, vital, and a deep red color. With minimal manipulation, granulation tissue bleeds due to the marked capillary content. Once the granulation bed is continuous throughout the wound bed, a ring of light pink epithelial cells should be noted on the periphery of the wound. As contraction progresses, migration of the epithelium is evident across the wound surface. The wound takes on a stellate (star-like) appearance as contraction progresses and lines of tension are evident. Once the wound can contract no further, the remainder of the healing process occurs by epithelialization, leaving a variably-sized, fragile epithelial scar.

Any delay in wound progression should cause a clinician to pause, and consider factors integral or supportive to wound healing. These factors can be intrinsic to the wound, and include infection, persistent inflammation, presence of foreign material, lack of appropriate blood supply to support healing, repeated trauma to healing tissue, and the presence of cancer, to name a few. These can also be patient factors, or those extrinsic to the wound, including poor nutritional status, systemic disease, and congenital abnormalities of wound healing.

Wounds should advance in an orderly fashion towards healing. It is important to have a plan in mind of how a wound should appear each day. Any delay in the predictable maturation of a healing wound often indicates a problem.

Wounds have been classified according to various criteria. The scheme that I find most helpful is dictated by the condition or character of the wound. A **clean** wound is a surgical incision created under sterile conditions. These wounds do not include entry into contaminated areas (gastrointestinal, respiratory, genitourinary). A **clean-contaminated** wound does enter one of

the aforementioned body systems without major contamination. In addition, clean wounds with a minor breach of sterile technique fall into this category.

Most wounds acquired via injury fall into the last two categories, **contaminated** and **dirty/infected**. Contaminated wounds include open traumatic wounds, and surgical wounds with major breaks in sterile technique. Dirty/infected wounds include old traumatic wounds in which greater than 10^5 organisms per gram of tissue are present.

Removal of devitalized and necrotic tissue is an important part of wound management. It may take several days for a wound to fully “declare” itself, and serial debridement may be necessary. Removal of dead and dying tissue as well as foreign material in the wound decreases inflammation, and speeds wound healing. In addition, the risk of infection increases with debridement delay. As soon as the patient is stable for surgery, debridement should be accomplished.

There are few indications for wet-to-dry bandages in veterinary medicine, except in heavily contaminated wounds such as shear wounds. A wide-meshed gauze can trap particulate matter and remove it from the wound surface at the time of bandage change. It is important to recognize that healing tissue is removed just as effectively – therefore, these bandages are only indicated in the earliest stages of wound management. Once granulation tissue begins to appear, this is no longer an appropriate means of debridement.

It is essential to cover wounds, keeping their surface moist and providing an optimal environment for healing to occur. The **primary** layer of a bandage is that in contact with the wound surface. A myriad of products are available, and many contain antimicrobial agents. The contact layer is chosen based on the stage of wound healing, the degree of contamination of the wound, and amount of exudate coming from the wound. The **secondary** layer of a bandage is the absorbent layer that draws and removes wound exudate. It should be applied thick enough that strikethrough is not anticipated prior to the next scheduled bandage change. This will maintain a capillary effect across the bandage. The **tertiary** layer simply holds the bandage together and protects it from the environment.

A variety of commercial products are available, and should be used according to the manufacturer’s instructions. These range from films, which provide a gas permeable but relatively fluid impermeable environment to hydrogels and alginates, designed to absorb large amounts of wound fluid. Topical antimicrobials are also available, such as silver and tri-peptide copper complex. Triple antibiotic ointment and gentamycin ointments may also be used sparingly, although petrolatum may inhibit epithelialization. Sugar and honey are also making a comeback as topical wound therapeutics, and can be combined with aminoglycosides or betadine for antimicrobial properties. It is important to recognize that there is no “magic bullet” for wound healing. Wound dressings may speed healing by a day or two over untreated wounds, and they are not a cure for poor wound management.

If at any time, wound healing is not progressing in an orderly, anticipated fashion, the wound should be cultured. This process involves flushing and cleaning the surface of the wound aggressively, and obtaining a piece of granulation tissue in a sterile fashion. It is essential to

culture tissue, rather than exudate, as the offending organism is deep-seated. Both aerobes and anaerobes are suspect.

APPROACH TO WOUND CLOSURE

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The choice of closure depends on the classification of the wound, as well as the size and location of the defect. It is imperative that infection be controlled, and contamination be minimized prior to wound closure.

In general, primary wound closure is reserved for wounds created under aseptic conditions, or those which were created sharply, with minimal tissue dissection. Delayed primary closure describes closure in 3-5 days if no evidence of necrosis or infection is noted. Secondary closure is closure performed after the formation of granulation tissue. This can be accomplished either by closing the skin and granulation tissue, or by excising the granulation tissue and closing the skin.

Second intention healing is the process by which wounds heal by contraction and epithelialization. This requires appropriate wound management during the healing period, and may result in a less cosmetic result than other closure methods. While economical, some wounds are too large to heal, and result in a fragile epithelial scar. Others may heal by contracture, resulting in loss of limb function. A good rule of thumb when determining whether a wound can or will heal by second intention is to pinch the skin together. If the skin edges do not meet, the wound is unlikely to heal by contraction alone.

Methods to increase skin stretch can be employed prior to closure, or during the closure process. While implantable skin stretchers are commercially available, they are often not feasible for veterinary patients. Tie-over bandages are an excellent means to recruit nearby skin during the wound preparation process. Skin has a remarkable ability to adjust to and lessen tensile forces over 12-48 hours. Application and adjustment of tie-over bandages gradually increases the amount of recruited skin around the wound for eventual closure. Similarly, adjustable mattress sutures can be placed intradermally, and used to gradually bring wound edges closer together. Walking sutures can be placed to combat tension across stretched skin; and releasing incisions can be employed to relieve excess tension in areas of limited soft tissue coverage.

When local tissue is unavailable to close a defect, flaps can be used to mobilize nearby (reliant on the subdermal plexus) or distant skin (with direct cutaneous arterial blood supply) to cover a defect. Subdermal plexus flaps are either advanced, or rotated into position to fill a defect, and circulation is collateral. It is important to plan these flaps keeping in mind lines of tension over the skin. The base of the flap should be slightly wider than the flap to preserve blood supply. It is also important to recognize that these flaps are limited in the distance they can cover. Flap necrosis will occur if the metabolic demands of the tissue cannot be met by the subdermal blood supply.

Axial pattern flaps are a much better choice when larger areas require coverage. These flaps have well-defined parameters, which are dependent on a cutaneous artery and nerve branch. Axial pattern flaps are not difficult to employ; however, it is crucial that the animal is positioned properly, as to accurately estimate where the cutaneous vasculature lies. The boundaries of the

flap are then drawn on the animal with a sterile marking pen, to ensure adherence to the known borders of the flap. The flap is then elevated below the level of the cutaneous musculature, and used to cover the defect. Care is exercised at the base of the flap, as if the cutaneous vasculature is damaged, the flap will uniformly fail.

Other complications associated with any type of flap are seroma formation, infection, dehiscence, and either partial or full necrosis. Adherence to careful surgical technique is expected to minimize these complications. The edge of flaps should be manipulated with stay sutures, or towel clamps, as to protect them from crush artifact of tissue forcep use. Similarly, the less electrocautery used at flap edges, the better. A good rule of thumb is if the thought of using a drain crosses your mind, then use a drain! Principles of placement should be followed – the drain should be placed in an aseptic manner, and exit in a dependent fashion, at a site away from the wound closure. In addition, the drain should not lie directly below the suture line. All drain exits should be bandaged, to absorb exudate and protect the wound from external contamination.

Skin without its own blood supply, in the form of a graft, (either partial or full thickness) can also be harvested to cover defects. In order for grafts to survive, immobilization of the wound is key. All exposed bone, tendon and ligament must be covered with granulation tissue for the graft to “take”. For the first several days, nutrition of the graft is through fluid from the wound bed, known as plasmatic imbibition. Then, capillaries invade the graft, in a process known as inosculation. Once this process begins, the graft takes on a more healthy, reddish hue, and is typically quite incorporated by 7-10 days post-placement. Grafts can be full, or partial thickness. Full thickness grafts are more robust, and heal with a more cosmetic appearance, while partial thickness grafts tend to take more easily, with a less satisfactory visual result.

PERINEAL SURGERY

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Perineal Hernias

The pelvic diaphragm serves to separate the abdominal contents from the perineal area. It consists of the external anal sphincter, levator ani and coccygeus muscles, along with the sacrotuberous ligaments (absent in the cat). These muscles serve to assist with tail movement as well as resist forces from abdominal contents during periods of increased abdominal pressure.

86-95% of dogs affected by perineal herniation are intact male dogs, suggesting a hormonal influence on the condition. Other considerations that have been proposed are neurogenic muscular atrophy, close cropping of the tail, as well as any condition that may lead to chronic increases in intra-abdominal pressure (intractable cough, severe GI disease, etc...). Regardless of the etiology of perineal herniation, most affected dogs will become clinical for the disease with time. Typical signs include straining to defecate, dyschezia, hematochezia and varying degrees of perineal swelling. While affected animals may show progressive signs over the course of years, it is important to recognize that perineal hernias can have acute, life-threatening emergency presentations. Acute urethral obstruction and urinary bladder entrapment/necrosis can occur, as can entrapment and strangulation of other abdominal contents, most notably the small intestine.

Conservative management of perineal hernia often includes diet modification and stool softeners. Enemas with deobstipation may be intermittently necessary. As 25-50% of dogs with perineal hernias have concurrent prostatic disease that may be contributing to caudal pressure, castration is always indicated. 47-66% of hernias are unilateral, but bilateral disease is common. About 10% of unilateral cases will herniate the contralateral side within months to years of surgery, more commonly in intact males, further strengthening the argument for castration.

Should life-threatening consequences ensue, aggressive fluid therapy, stabilization and emergency abdominal exploration may be necessary in addition to hernia repair. In the case of urinary bladder entrapment, decompression during the stabilization process is paramount to assist in treatment of hyperkalemia (if present) and for urinary diversion in the rare case of uroperitoneum. If an indwelling urinary catheter cannot be passed easily, decompression via cystocentesis with ultrasound guidance is indicated, followed by catheter placement.

Traditional herniorrhaphy consists of placement of interrupted mattress sutures between the external anal sphincter and surrounding available musculature. In the dog, the sacrotuberous ligament is often employed, and may lead to sciatic nerve entrapment. Should this occur, animals recover from anesthesia in excruciating, intractable pain, necessitating additional surgery. The sciatic nerve is explored via a caudal approach to the coxofemoral joint, and the offending suture is removed. Other more common complications of this technique include seroma formation, dehiscence, reherniation and incisional infection.

As there is often a paucity of tissue with which to perform traditional repair, other methods of herniorrhaphy involve transposition of additional musculature into the perineal area to fill the

pelvic diaphragmatic defect. Techniques described include use of the internal obturator muscle, the superficial gluteal muscle, and the semitendinosus muscle. Each of these techniques varies with respect to invasiveness, ease of performance, and complication rate.

Various materials have been used as sole products for hernia repair, or to augment muscular transposition. These include fascia lata, porcine small intestinal submucosa, and polypropylene mesh, among others. Additionally, supportive intra-abdominal procedures can be employed. The object of these procedures is to help prevent caudal displacement of abdominal organs and pressure on the hernia repair. Cystopexy and colopexy are the most common procedures performed, where the urinary bladder and descending colon are scarified and sutured to the abdominal wall. The resultant adhesion formation, prevents caudal displacement. Vas deferentopexy has also been described.

Regardless of the technique chosen for herniorrhaphy, some general tricks to assist in hernia reduction are a Trendelenburg position, where the head is directed downward. This helps keep the abdominal structures within the abdominal cavity through gravity. Also, a gauze sponge rolled and held with Allis tissue forceps can be placed through the hernia defect to help keep the contents from the surgical field. A purse string suture is essential in the rectum to prevent fecal contamination of the surgical site, and any stool softeners should be discontinued a few days prior to surgery to decrease fecal contamination.

Post-operative management of these cases includes a low-residue diet, restricted activity levels, and treatment of other diseases that may increase intraabdominal pressure. The animal should not be allowed to lick or chew the incision, or “scoot” it along the ground. Complications are seen in up to 45% of cases with internal obturator transposition, and 60% of cases where traditional herniorrhaphy is employed. Many are self-limiting, including swelling, seroma formation and pain. Infection may also be seen.

Certainly the most concerning complications include sciatic nerve damage, fecal incontinence and reherniation. Avoiding the use of the sacrotuberous ligament in hernia repair can prevent sciatic nerve damage. Fecal incontinence can be temporary or permanent, and results from damage to the caudal rectal branch of the pudendal nerve, external anal sphincter or both. Unilateral nerve damage resolves over time as the contralateral caudal rectal nerve reinnervates the external anal sphincter muscle. However, bilateral damage to these nerves causes permanent incontinence (reported in <15% of cases).

Dysuria has also been reported, especially after urinary bladder entrapment and resultant atony. This may have a higher incidence in animals undergoing cystopexy or vas deferentopexy. Tenesmus is also relatively frequent and concerning after surgery, since straining to defecate places immense pressure on the repair, as well as the contralateral pelvic diaphragm.

Reherniation occurs in 10-50% of dogs undergoing traditional herniorrhaphy, and up to 36% of dogs after internal obturator transposition. However, some studies show a recurrence rate as low as 8%. Recurrence rates for inexperienced surgeons far exceeds that of the seasoned surgeons (70% vs 10%). As with most surgeries, the first chance at repair yields the best chance of success. A 90% success rate has been reported in one recent study that augmented internal

obturator transposition with intraabdominal techniques,² and a review of 59 cases operated by experienced surgeons described a reherniation rate of 12% when mesh was used to augment the surgical repair.³ These results suggest that a more aggressive approach during initial surgery may decrease reherniation rates, and need for additional surgery.

Anal Sac Disease

Anal sacs are paired diverticula located between the internal and external sphincter muscles, and it is estimated that up to 12% of veterinary visits involve these structures.⁴ Each empties via a single duct, which can become impacted. Apocrine glands open into the glands themselves, with sebaceous glands opening into the ducts. Arterial supply is via branches of the internal pudendal and caudal gluteal arteries, and innervation via the perineal nerve.⁴

Anal gland secretions consist of desquamated keratinocytes, and material from sebaceous and apocrine glands. Various species of bacteria and yeast are also normal inhabitants.⁵ Large amounts of amorphous basophilic debris is also noted on stained slides. One report indicated that neutrophils, when present are non-degenerative, whereas erythrocytes should be absent.⁵ A second report, however, described the majority of neutrophils in normal animals as degenerate.⁶ A third study concluded that cytology is not useful in distinguishing between normal and diseased anal glands.⁷ It is agreed that color and consistency of anal sac secretions are variable, and are not valuable predictors of anal sac disease.⁵⁻⁷ Further confounding assessment of anal gland secretions is the fact that bacterial species found in normal and diseased anal sacs tend to be the same.⁵⁻⁷ Therefore, it is difficult to use cytology or bacteriologic culture to confirm anal sac disease, or predict animals at risk of impaction of abscessation.

Anal sacculitis and impaction are common conditions, and are diagnosed via clinical signs and digital rectal examination. The glands are often enlarged and painful, with blood contamination of the expressed contents.⁴ numerous predisposing factors, such as the size of the animal, obesity, skin disease, inflammatory intestinal disease, diet and poor activity level, have been proposed.⁸ Impaction and abscessation can be a sequellae to this process in which the gland swells, is extremely painful, and can fistulate through the skin. Often, dogs with anal gland abscesses are systemically ill.

In order to determine whether anal sacs are diseased, and to treat anal gland impaction, a digital rectal examination is performed. Each gland is evaluated and digitally manipulated to evacuate the contents. Internal expression is preferred, as it allows complete removal of contents. If the animal is painful, sedatives may be required for this procedure. Local flushing and infusion of the glands with a chlorhexidine solution or otic preparation containing topical antibiotics +/- steroids is often employed. Parenteral therapy is indicated only with signs of systemic illness.⁴ It is essential to completely evacuate the gland contents to examine the gland itself for masses that can be consistent with anal gland adenocarcinoma.

Anal saccullectomy is recommended in cases of recurrent anal sacculitis, abscessation or neoplasia. Both closed and open techniques (in which the gland is entered during removal) have been described. The closed technique results in less bacterial contamination of the surgical site.⁸

Briefly, a small incision is made over the anal sac, which can be augmented with water-soluble lubricant instilled into the gland, paraffin wax, or even a small foley catheter.⁹ The dissection is continued very close to the gland until it can be removed, ligating the duct at its attachment to the rectal opening. The surgical site is then copiously lavaged and closed with fine absorbable monofilament suture material. In the open technique, small scissors are introduced through the anal gland duct and an incision is made, essentially filleting the duct and gland open, which are then dissected.

Complications of anal saccullectomy include infection, abscessation with fistula formation, and fecal incontinence.⁸ Incontinence is rare, and is due to damage to the caudal rectal nerves bilaterally with aggressive dissection, or through removal of >50% of the anal sphincter muscle.⁸ In a retrospective study comparing the open and closed technique for non-neoplastic anal sac disease in dogs, the open technique was associated with a greater complication rate.¹⁰ 14 of 95 dogs had long term complications, including stricture formation, fecal incontinence and fistulation.

Anal gland adenocarcinoma should be considered with any mass located in the vicinity of the anal sac, and excision is a standard part of multimodal therapy. Prognosis is better for tumors that are less than 10cm at the time of diagnosis.¹¹⁻¹² Hypercalcemia was noted in 27% of dogs, with confirmed pulmonary metastasis in 8/95. The median survival time was significantly shorter for dogs with pulmonary metastasis.¹¹ In another study of apocrine gland adenocarcinomas, four negative prognostic indicators were determined: lack of therapy, distant metastasis, local lymph node metastasis and tumor size.¹²

Median survival time is approximately 18 months, and surgical intervention provides a survival advantage. Other therapies that can be employed include local radiation therapy and chemotherapy for advanced disease. As very few animals are presented with noticeable anal sac masses, complete evaluation including rectal examination must be performed yearly to diagnose adenocarcinoma in its early stages. Similarly, any animal presenting with hypercalcemia should have a digital rectal exam.

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PRINCIPLES OF CUTANEOUS ONCOLOGIC SURGERY (APPROACH TO LUMPS AND BUMPS)

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Preoperative planning is perhaps the most important aspect of oncologic surgery. Advances in imaging, anesthesia, emergency and critical care have been essential in moving the discipline of veterinary oncology forward, allowing more radical procedures to be performed. Complete perioperative staging can yield information critical to estimating prognosis for a given animal, further directing surgical recommendations.¹

Surgical biopsy is an important and often overlooked area in veterinary oncology. It is accepted that the first attempt at surgery often gives the best chance of operative success.^{2,7} Knowledge of tumor type not only helps predict biologic behavior, but also is essential in guiding the surgeon with respect to the extent of surgical excision. With respect to soft tissue sarcomas, three centimeter margins are recommended to prevent local recurrence, whereas mast cell tumors can be more conservative at two centimeter margins and one fascial plane deep.³ Current recommendations for feline vaccine-associated sarcomas are five centimeters around the tumor.⁴ Another study showed similar results for recurrence with clean and close surgical margins, emphasizing the need for proper planning prior to surgery.⁵

Biopsy can be extremely useful not only to determine a diagnosis, but degree of local invasion, expected metastatic activity, and other biologic behavior of the tumor in question. This information assists the surgeon in determining the best plan for surgical excision, and provides information for the owner regarding extent of surgery and an expectation following the procedure.^{1,2,6} Biopsy also ensures that other similarly efficacious treatment modalities, if available, can be explored with the owner prior to surgery.

Prior to discussion of biopsy, it is important to perform a complete physical examination, and staging of the patient. This will allow discovery of comorbid conditions, paraneoplastic syndromes and potential metastatic disease: all of which may alter the surgical plan.⁶ Typical staging includes a complete blood count, serum chemistry panel, three view thoracic radiographs, fine needle aspirate of the tumor and local lymph nodes, +/- abdominal imaging.

Fine needle aspirates can yield a definitive diagnosis in some instances, such as with mast cell tumors and lymphosarcoma. In the case of equivocal results, biopsy is recommended for further classification. Biopsy can be of needle type, incisional, or excisional. Regardless of the type of biopsy chosen, all instruments and suture material should be changed when more than one mass is biopsied.^{2,6} Certain masses may not require biopsy, such as with solitary splenic or lung masses, as the surgical treatment is not altered. Similarly, if biopsy would carry the same risks as surgical excision, excision is planned.

In general, the biopsy technique chosen should be simple to perform, yield sufficient tissue for histologic diagnosis, with few complications. Electrocautery is used minimally on the sample submitted for biopsy. Proper preservation techniques (1:10 volume of tissue to formalin) must be known and followed, and the sample submitted to a veterinary pathologist for definitive diagnosis.

Needle biopsy, such as with a Tru Cut is superior to fine needle aspiration, but inferior to incisional or excisional biopsy. A small core of tissue is removed, which then is manipulated carefully to ensure that the architecture of the sampled tissue is unchanged. Excessive trauma to the tissue from forceps or other instrumentation may yield the sample as non-diagnostic. Multiple samples are collected to improve diagnostic accuracy. It is also important to map the location where the biopsies were collected, as all biopsy tracts must be removed with the tumor.

Incisional biopsy is removal of a portion of the tumor, along with a margin of healthy tissue. Incisional biopsy should be avoided in areas of necrosis or ulceration to prevent collection of a non-diagnostic sample. Excisional biopsy describes removal of the entire tumor, often without knowledge of appropriate margins. When performing incisional and excisional biopsies, scar location and orientation is of paramount importance, and is often overlooked. When complete margins are not obtained, by definition, the remaining mass and previous scar are considered contaminated. Appropriate margins must now be taken around the mass as well as the scar. In some clinical scenarios, this results in much more aggressive surgery than would be required with preoperative forethought.

After surgical biopsy, drains are never used. Drains act to facilitate removal not only of fluid and debris, but cancer cells that can then seed the drain tract. Again, if further surgery is pursued, the drain tract is also considered contaminated.

When planning for surgical excision of a tumor, consider first the diagnosis if known, and ensure that appropriate staging has been performed. Next, confirm that the histologic diagnosis matches the clinical course seen in the patient. Then, consider tumor location, and whether advanced imaging (CT/MRI) would be beneficial in determining the extent of tumor invasion prior to surgical resection.⁷ In contrast, if adjunctive therapy is available, marginal excision may be chosen, where microscopic disease is left behind in the wound bed to be addressed by multimodal therapy.

Radical resection is considered when the surgery has curative intent. The skill level of the surgeon is important if the mass is adjacent to important neurovascular structures, or if an advanced knowledge of anatomy is essential for success. The excision is planned and marked out on the patient prior to incision. Separate instruments are used for excision of the mass and closure. During excision, major vascular structures are ligated early, to prevent spread of tumor microthrombi through the vasculature during tumor manipulation. Attention is given to meticulous dissection and accurate hemostasis. Gloves should be changed between removal and closure, and the wound bed copiously lavaged. Closure is with appropriate gauge monofilament absorbable suture.

Once removed, the tissue is marked for orientation. This can be performed with sutures or staples. Ink is available to coat the entire lateral and deep margins to give the pathologist a reliable indicator of the cut edge. Again, a 1:10 ratio of tissue to formalin is a must, and since formalin penetrates maximally to 1 cm, the tissue should be incised at regular intervals to allow adequate fixation. Special handling (as for eye, nerve or brain tissue) if necessary, must be

known prior to surgery, so that appropriate samples are put aside and treated appropriately prior to desiccation.

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MEDICAL AND SURGICAL TREATMENT OF TRACHEAL COLLAPSE

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Diagnosis and Medical Management of Collapsing Trachea

Diagnosis:

“Tracheal collapse” is a blanket diagnosis that covers a myriad of structural anomalies of the conductive airways. Collapse can be ventrodorsal or lateral, generalized or focal, can involve neither, one or both principal bronchi, etc... An underlying primary cartilage defect is suspected, along with mitigating factors that can precipitate or exacerbate clinical signs. Due to the dynamic nature of this disease and its unique presentation in each patient, the options for medical management are vast and the criteria for surgical intervention are often indistinct.

Tracheal collapse is a progressive, irreversible condition of the lower airways commonly associated with a “goose honk” cough and varying degrees of airway obstruction. Early affliction is characterized by laxity of the trachealis muscle, which progresses to weakness of the cartilagenous rings. The collapse of the cartilage infrastructure ultimately leads to obliteration of the tracheal lumen, usually in a dorsoventral manner. Tracheal collapse is a condition that normally affects toy and small breed dogs, specifically Yorkshire terriers, Miniature Poodles, Pomeranians, Chihuahuas and Pugs. A sex predilection has not been described; however, there is an age predilection for middle-aged dogs. As many as 25% of affected dogs are symptomatic by approximately 6 months,¹ and some studies have shown that younger dogs are more severely affected than those presented in middle age. It is hypothesized that a congenital component is present in many affected dogs, but that other external factors such as obesity, environmental allergens, cigarette smoke, and Bordetellosis exacerbate clinical signs. This has been described as a congenital tendency with “initiating” factors, and of 100 cases studied, 55% had one or more co-existing diseases.¹

While the above signalment is typical, tracheal collapse has been reported in both large breed dogs and a cat.

Histopathologic examination of the tracheal cartilage in affected animals is found to be hypocellular with a reduction in glycoprotein and glycosaminoglycan, allowing for decreased retention of water within the matrix. The decreased water retention leads to an increased compliance and decreased rigidity of the tracheal cartilage. Decreased chondroitin sulfate and calcium may also allow for replacement of hyaline cartilage with collagen and fibrocartilage, enhancing the weakness of affected tracheal rings. In collapsed tracheas, width to height ratios can approach 1.5:1 to 4:1 depending on severity and location within the trachea. A vicious cycle of cough and perpetual inflammation ultimately leads to loss of normal tracheal epithelium and replacement with a fibrinous membrane epithelium, followed by formation of squamous metaplasia and a reduction of ciliated cells with secretion of increasingly viscous mucus. Pulmonary hypertension with right ventricular enlargement and cor pulmonale has been reported in dogs with tracheal collapse.

There are various modalities used for the diagnosis of tracheal collapse, and the intensiveness of the tests pursued is based on the likelihood of adjunctive therapy. A thorough physical

examination is often an excellent means of diagnosis, in that an elicitable cough may be present on tracheal palpation, or the cervical tracheal rings may be flattened. Similarly, in dogs with intrathoracic collapse, careful auscultation may confirm narrowing of the airway and wheezing as the patient exhales. At our tertiary referral center, dogs rarely present for mild tracheal collapse. Therefore, we attempt to educate the owners of susceptible breeds when they present for other problems, such as luxating patellas, portosystemic vascular anomalies, routine ovariohysterectomy or castration, etc...

Radiographs are often used to confirm a diagnosis of tracheal collapse, as well as to rule-out other etiologies of cough. It is best to capture both an inspiratory and expiratory film to allow examination of the cervical and thoracic trachea when most likely to collapse. However, radiographs represent a static moment in time, and can miss a diagnosis of tracheal collapse.² When available, fluoroscopy is also an excellent means of diagnosing tracheal collapse, as the unsedated patient can be observed in real time. Often, the most accurate extent of collapse can be seen when a cough is elicited.

Tracheobronchoscopy has emerged as the gold standard test for the evaluation of tracheal collapse.³ The extent of collapse can be accurately graded and measured, and the tracheobronchial mucosa can be directly visualized. Advancement of the scope into the principal bronchi and beyond yields information regarding patency of the smaller airways, and sampling of these areas either with cytology brushes or bronchoalveolar lavage can be accomplished. The biggest limitation of this technique is the need for general anesthesia. While uncommon, occasionally an anesthetic episode will cause acute exacerbation of tracheal collapse, rendering extubation impossible. This is discussed with the owners prior to tracheoscopy, to allow decision-making prior to a period of crisis.

Medical Management:

The cornerstone of therapy for dogs with tracheal collapse is aggressive medical management.¹ Weight loss is an important component of this process, as excess fat deposition in the thorax can further impinge on the trachea, and obese dogs have more difficulty with thoracic excursions. Exercise may be prohibitive in severely affected patients until a measurable proportion of their weight is lost, so a calorie-restricted diet is a must. A prescription weight loss diet is ideal for these patients, as it will facilitate the most weight loss in the shortest amount of time. It is worthwhile to spend a great deal of time designing a weight loss and exercise regime for these dogs and to encourage the owner to be an active participant in the health of their pet, as compliance is often difficult. Another advantage of using a prescription diet is that the owner can be taught to think of the diet as a drug, further emphasizing the importance of calorie restriction.

Environmental modification can also be extremely helpful in controlling the clinical signs of tracheal collapse. Dogs should be walked with a harness, rather than a collar to minimize pressure on the neck. Similarly, slow controlled walks are preferred to bursts of vigorous activity. This will encourage weight loss without precipitating cough from increased airway pressure. Exposure to airway irritants should be minimized – common culprits include cigarette

smoke and heavily perfumed products such as air fresheners, candles and carpet treatments. Airway humidification can also diminish clinical signs.

Treatment of concurrent diseases is also recommended, and can be beneficial in reducing or controlling clinical signs. Left atrial enlargement from cardiac disease places pressure on the carina, and can exacerbate bronchial collapse. Untreated endocrinopathies may preclude weight loss and predispose to infection.

Pharmacologic management:

Numerous drugs have been used for the management of tracheal collapse, and a regime must be tailored to each individual patient. Just as some animals respond preferentially to one NSAID over another for the treatment of arthritis, experimentation with multiple drugs in the same class may be necessary to determine which is best suited for the dog. Drugs that are considered “mainstays” of therapy include antitussives, sedatives and bronchodilators. Short courses of corticosteroids are also beneficial in breaking the cycle of inflammation. Chondroprotectants have also been used, but their benefit in the etiology of tracheal collapse has not been definitively demonstrated.

Narcotic medications are potent cough suppressants. Hydrocodone, butorphanol and codeine are commonly used. These drugs are given to effect, and require dose and interval modification over time. Of these, butorphanol is the most potent cough suppressant (100 times greater than codeine), but its short duration of action makes hydrocodone a more suitable choice for most patients. Dextromethorphan may also be used, as some owners appreciate less of a sedative effect; however potency is equal to that of codeine.

Sedatives can be used intermittently, especially if a particularly stressful situation or period of excitement is expected. However, in severely affected animals, chronic low-level use may be indicated.

Bronchodilators may be helpful, as their use may result in an overall decrease in intrathoracic pressure during expiration. In addition, mucociliary clearance rate may improve with the use of these medications.

Although anticholinergics have been used in the past to decrease cough from irritation, they are generally contraindicated in cases of tracheal collapse. These drugs can induce bronchoconstriction, dry airway secretions and slow ciliary motion.

Intermittent use of antibiotics may be necessary, especially if there is an acute exacerbation in clinical signs. Ideally, tracheobronchoscopy is used in order to collect airway samples for cytology and bacterial culture. Many of these patients have been empirically treated with antibiotics, and resistant strains of *Bordetella* are particularly problematic. *Mycoplasma* may also be isolated, and appropriate treatment of these infections may result in substantial improvement, further delaying surgical intervention.

Similarly, short courses of corticosteroids at anti-inflammatory doses may be used occasionally in an attempt to quell the cycle of inflammation. Routine use should be avoided if possible, to prevent iatrogenic Cushing's disease, weight gain, and decreased resistance to infection.

Surgical Management of Collapsing Trachea

Controversy continues to exist regarding the timing of surgical intervention, as well as the preferred means of tracheal support (intraluminal, extraluminal or both). Client education is extremely important, as life-threatening complications may be experienced with any of these techniques. In addition, these dogs will continue to cough after surgery. Therefore, if the chief complaint is cough, it is likely that the client will be less than satisfied with the outcome. We typically reserve surgical options for those animals that have failed appropriate medical management, or have acutely begun to experience life-threatening cyanotic episodes that have required hospitalization.

In my practice, self-expanding veterinary nitinol stents are employed for intrathoracic collapse, and extraluminal prosthetic rings are used for isolated cervical collapse. Extraluminal rings are available commercially, or can be constructed from polypropylene syringe cases. A midline cervical approach is used, and the prostheses are placed no more than 2-3 tracheal rings apart, to negate the possibility of collapse between the supported areas. It is essential that sutures are placed 360 degrees around the trachea, and that the trachealis dorsalis muscle is engaged to maintain luminal patency. Traction is used to mobilize the trachea, and draw as much of the thoracic trachea into the surgical field as possible. We place a steel suture or hemoclip at the level of the last prosthetic ring to confirm location on post-operative radiographs, and find that the thoracic inlet can be spanned to the level of the first or second rib on a lateral film. Often, smaller rings are employed as the cervicotracheal junction is reached, and the trachea narrows. Complications with this technique are minimized when the modified approach is used, but tend to be serious and include laryngeal paralysis, tracheal necrosis, pneumomediastinum and pneumothorax.⁴

Intraluminal stenting is an attractive option, as placement is rapid, improvement is immediate, laryngeal function is not adversely affected, and incisional complications are negated.⁵⁻⁷ Unfortunately, at our institution approximately 10% of stents have fractured after placement, resulting in a poor outcome and death.⁶ Other complications include respiratory infections, tracheitis and excessive granulation tissue formation. The cost of stent placement is also double that of prosthetic rings, and may be cost-prohibitive for many owners.

Stent sizing and placement: Appropriate stent sizing is crucial. The description of sizing and placement in these notes is my personal preference; there are modifications of these techniques that are used at other institutions. Accurate tracheal length and width is necessary to determine stent size. We obtain lateral radiographs under general anesthesia, and place the endotracheal tube cuff as close to the larynx as possible. A positive pressure breath of 20-25 cm of water is maintained as the films are taken, effectively distending the tracheal lumen. A calibrated esophageal marker is also used to account for magnification. Multiple measurements of tracheal width are collected from the entire length of the trachea. The stent chosen should exceed the

width of the trachea at its widest point to prevent dislodgement during cough. Due to the discrepancy in width between the cervical and thoracic trachea, it is possible that the stent chosen will not expand fully in some areas. The manufacturer's shortening charts should be used to ensure that the chosen stent will not be longer than the trachea.

I prefer to place the stent under direct visualization using a rigid scope. I believe that this allows the most accurate placement of the distal portion of the stent. It is important to maintain the scope at a location in which the carina is easily observed, but that complete expansion of the distal portion of the stent is not inhibited. After the stent is deployed, the scope can be gently removed from the trachea and reinserted in the stent lumen to document placement.

Post-Operative Management:

The main concern after both extraluminal ring placement and stent placement is the control of airway inflammation and the prevention of cough. This is especially critical for dogs with stents. Under the continual outward pressure exerted by the expanded stent, the tracheal mucosa is expected to migrate over the implant, re-instituting the mucociliary escalatory apparatus. It is hypothesized that fracture incidence may be reduced if this process occurs uneventfully. Each time the animal coughs, the fragile mucosa is disrupted and the embedding process is delayed. Cough will also fatigue the material of the stent, making fracture more likely. Typically, our post-operative protocol consists of routine acetylpromazine for two weeks after the procedure and hydrocodone as needed indefinitely to prevent cough. A two-week course of amoxicillin/clavulanate is prescribed, as well as a tapering dose of anti-inflammatory prednisone. Continued medical management is typically required, and again is tailored for the individual patient.

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COMMON QUESTIONS FROM RDVMS REGARDING THE GASTROINTESTINAL AND IMMUNE SYSTEMS IN DOGS AND CATS

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What is this new lipase?

Pancreatitis in the dog is relatively common but can be frustrating to definitively diagnosis. History, clinical signs and physical examination findings lead to the differential diagnosis; however, the presentation can be nonspecific and variable. A diagnosis is generally based on the combination of these signs plus clinicopathological and imaging findings.

Imaging of the pancreas depends on operator experience, equipment and degree of inflammation in the pancreas and surrounding tissues. The specificity of abdominal ultrasonography was only 68% in one study.

The original serum lipase was a catalytic assay that used a 1, 2 diglyceride assay. This test was unreliable for the diagnosis of pancreatitis because of low sensitivity and specificity. Lipase is produced by tissues other than the pancreas. Serum amylase concentration also lacks sensitivity and specificity.

The canine pancreatic specific lipase is available as a SNAP cPL and as a quantitative test Spec cPL. Sensitivity of Spec cPL concentrations vary for mild pancreatitis (21%), moderate to severe pancreatitis (71%), and pancreatitis overall (64–94%). Currently, serum cPL concentration is considered the most specific serum biomarker for the diagnosis of pancreatitis in dogs. Specificities range from 71–100%.

The new lipase test, 1,2-o-dilauryl-rac-glycero-3-glutaric acid-(6'-methylresorufin) ester (DGGR) assay, was validated in dogs in 2005. The DGGR lipase test was recently compared to the Spec cPL and found to have high agreement. The DGGR lipase assay may be less expensive and have a faster turn-around time than the Spec cPL depending on the laboratory used.

It must be remembered that any lipase test is not 100% sensitive nor specific and the only true method of diagnosis is histopathology. As an example, in a study that we completed at the University of Tennessee, dogs with HAC but without clinical pancreatitis were more likely to have abnormal SPEC cPL concentrations and SNAP cPL results than dogs with normal ACTH stimulation test results. Based on this study, abnormal cPL results should be interpreted with caution in dogs with HAC in the absence of clinical signs of pancreatitis. Further studies are warranted to determine the pathophysiology behind this association.

Is there a new parvovirus?

Canine parvovirus (CPV) was first reported in the late 1970's in North America (NA). This disease is caused by strains of CPV-2 (2, 2a, 2b, 2c). This is a single stranded DNA virus that has a higher mutation rate than other DNA viruses. The original strain CPV 2 mutated to develop the new strains CPV 2a, 2b and 2c over the next few years (1980, 1984, and 2000

respectively). The CPV-2b and 2c are the predominant strains in NA. CPV-2c differs from CPV-2b by a single amino acid. Tests and vaccinations work for both strains.

Acute infections can occur in any age dog but are more likely in puppies between 6 weeks and 6 months. There have been reports of outbreaks of CPV-2c infections in adult dogs (over 6 months of age). Severe cases may also be affected by co-infections of parasites and other viruses.

Initial testing for parvovirus includes fecal ELISA antigen tests and PCR methods. The point of care ELISA tests detect viral antigen in feces or rectal swabs but both false positives and negatives can occur. False negatives occur due to low concentrations of viral particles in the feces either due to decreased shedding in later stages of disease or dilutional effects of the diarrhea. False positives may occur following vaccination with modified live vaccines. PCR can also be false positive following vaccination. Virus isolation, hemagglutination and electron microscopy can be used to demonstrate infection but availability and turn-around time may be limiting for confirmation or group situations.

Treatment for CPV has not changed significantly over the years as it is supportive care. Fluid therapy and oncotic support are the mainstays of therapy. Antiemetics and antibiotics are added as needed. Management of nutrition has improved outcome. Immunotherapy with convalescent plasma has anecdotally been reported to help. Oseltamivir (Tamiflu) has been used as a treatment for influenza infection but has no theoretical basis for treatment. One study showed improved weight gain and maintenance of white blood cells in the oseltamivir treatment group compared to the control group. However, there was no significant difference in survival or clinical signs.

How do you approach chronic small bowel diarrhea?

Diarrhea can be described originating from small or large bowel or both. Small bowel diarrhea can be caused by nongastrointestinal disease such as endocrine, renal, liver, pancreatic, heart failure, metastatic cancer and toxins or drugs. A patient with small bowel diarrhea must be examined with this in mind and evaluated with basic laboratory and imaging assessment. It also must be remembered that there can be small bowel disease without diarrhea. Small bowel disease may have other clinical signs such as vomiting, weight loss, abdominal discomfort or altered appetite. Melena indicates blood has been digested in the small intestine – either through ingestion or localized gastrointestinal bleeding in the stomach or small intestine.

History and signalment will determine the initial approach. Differential diagnoses for a puppy may initially include foreign body (partial obstruction), infectious (parasitic, viral), diet, or even congenital abnormalities. Differential diagnoses for an old dog would more likely include neoplasia or inflammatory as primary. History is important. If several animals in a household are affected with diarrhea, the differential diagnoses are more likely to be infectious or dietary as a common cause.

Basic causes of diarrhea include malassimilation (maldigestion/malabsorption), secretory, osmotic, dysmotility or mixed. Dysmotility is seen with hyperthyroidism in cats. Secretory causes include bacterial enterotoxins or chemical toxins and possibly intestinal inflammation. Osmotic diarrhea results from unabsorbed products that lead to the osmotic draw. Malabsorption is a common cause of diarrhea and can be seen with many disorders (liver disease, bacterial overgrowth, lymphangiectasis, EPI, villous atrophy). A permeability or malabsorption diarrhea is also caused by intestinal inflammation or neoplastic infiltration.

Depending on the case, the approach to chronic diarrhea may vary. However, nongastrointestinal causes must be ruled out through blood work and imaging. Abdominal ultrasonography can evaluate bowel wall thickness and presence of masses. Fecal examination for parasites is absolutely necessary (direct smear, flotation, concentration methods). If these results are negative, deworming with a broad spectrum drug is still advised prior to more expensive and invasive diagnostic techniques.

Rectal cytology may reveal a large number of neutrophils which can indicate a bacterial problem. This test may help to decide if a bacterial culture would be useful. Rectal scraping may indicate the presence of histoplasmosis and the diagnosis is made!

Bacterial cultures are very confusing to interpret because of normal gut flora. If the animal is febrile, has an inflammatory leukogram, has hemorrhagic diarrhea and has increased neutrophils on rectal cytology, culture may be used (*Salmonella*, *Campylobacter*). Interpretation of the culture results must be carefully considered.

Common serum laboratory panels for GI include: TLI, PL, folate and cobalamin. TLI will rule in or out exocrine pancreatic insufficiency and the PL may rule out pancreatitis. Decreased serum folate concentration may indicate disease affecting proximal small intestine and increased concentration may indicate bacterial overgrowth. Serum cobalamin concentration is decreased with bacterial overgrowth or distal small intestinal disease. However, serum folate and cobalamin concentrations are insensitive and non-specific for detecting bacterial overgrowth. Also, bacterial overgrowth is better defined as antibiotic-responsive enteropathy (ARE). This syndrome is probably a result of increased bacteria in the upper small intestine and the resulting host response. The bacteria are not obligate pathogens and can be ones that are usually found in that area. The host response may be inflammatory or dysbiosis. ARE is difficult to definitely diagnose and empirical antibiotic therapy may be chosen as a means of diagnosis. However, antibiotic choice is difficult because any bacteria species can be present in the small intestine and the species may change over time. Tylosin is a popular antimicrobial choice. Also, metronidazole can eliminate many anaerobic bacteria and may also be immunomodulating. An antibiotic trial should be done for at least 2 – 3 weeks. The patient may respond to concurrently feeding a therapeutic diet trial.

If the patient can tolerate a therapeutic diet trial, this is worthwhile to rule out food sensitivity. There is no perfect trial diet but a careful examination of historic diets may help determine the initial elimination diet. A novel protein diet or a hydrolyzed diet are good initial choices. It must be stressed that during this trial, no other foods or treats can be given. This includes flavored heartworm or flea control products. This diet should be implemented for a minimum of

2 – 4 weeks. It is rare that longer periods are required as a trial. If the diet resolves the diarrhea, continue for another 3-4 weeks to ensure that it was not a transient improvement. It has been seen that up to 50% of case of chronic diarrhea will resolve with a diet change. Also we have seen that 50% of those case may be able to be changed back to the original diet with time.

Failure of response to diet change, antibiotic trial, or deworming can then indicate that endoscopy and biopsy are necessary. Some diseases will have normal biopsies including ARD, food intolerance, dietary indiscretion, toxic diarrheas, motility disorders, or systemic disease. Endoscopy allows visual examination of the gastrointestinal tract but does not include the mid jejunum and ileum. Colonoscopy may allow retrograde examination through the ileocolic valve into the ileum. Biopsies of the intestines should always be taken with chronic diarrhea and the result depends on the quality and quantity of samples obtained. Full thickness biopsies via surgical means may be necessary when endoscopic mucosal biopsies are non-diagnostic.

Protein losing enteropathy (PLE) in dogs can cause a chronic diarrhea and can result from inflammation, neoplasia, lymphangiectasis, infection or endoparasitism, and intussusception. There are breeds predisposed to PLE: Yorkshire terrier, soft-coated Wheaten terrier, Shar-Pei, Basenji, and Lundehund. These cases usually require biopsy for diagnosis.

Treatment of small bowel diarrhea depends on the underlying cause. If the diagnosis is inflammatory bowel disease or lymphocytic plasmacytic inflammation and diet, antibiotic, anthelmintic treatment does not help, then immunosuppression can be tried. This is usually corticosteroids, but other treatments may be useful. Cats can respond to chlorambucil.

The diagnostic approach to large bowel diarrhea is similar to small bowel. However, other infectious diseases such as trichuris, giardia, tritrichomonas (in cats), pythium and prototheca can occur in the large bowel. An inflammatory histiocytic ulcerative colitis or granulomatous colitis occurs most frequently in young Boxer dogs but can occur in other breeds. This disease has been linked to adherent and invasive E. coli (AIEC). This was diagnosed with biopsies that were tested by FISH (fluorescent in situ hybridization). Therefore if this disease is considered, mucosal biopsies can be submitted for histopathology, FISH test and also cultured for antimicrobial susceptibility. These cases have responded to long term enrofloxacin therapy (7 mg/kg/day for 9 – 10 weeks) but antimicrobial resistance is becoming common.

What about probiotics?

The intestinal microbiota is the new exciting hot topic. The complexities of this system are overwhelming and the research in human medicine is exploding. The inter-relationship between the microbiota and the host is fascinating. Because of this complexity it is not possible to give specific recommendations for the use of probiotics. For basic treatment, the probiotic must survive passage into the gut, adhere to the gut, and also proliferate. The probiotic does not survive long in the gut and therefore must be given on a regular basis. There is also variation between type and number of organism in different products.

Different products have been used in veterinary medicine and have been used with clinical improvement in chronic diarrhea. However, remember that these are bacterial organisms that can be susceptible or resistant to antibiotics administered to the patient.

Megacolon in cats – what to do?

Constipation and megacolon in cats is a challenge and must be fully evaluated to exclude underlying causes. With recurring cases, after history and basic physical examination, examination of the anorectal area and neurologic system is necessary. Basic laboratory assessment and abdominal and pelvic imaging are next to be evaluated. Laboratory results may indicate metabolic or endocrine problem. A rectal exam is difficult in a conscious cat and is reserved for heavy sedation or general anesthesia during removal impacted feces.

Neural deficits may reveal a neurologic cause such as neuromuscular dysfunction. Other mechanical causes can include pelvic stenosis, colonic neoplasia or perineal hernia. When all is ruled out, idiopathic constipation is diagnosed and medical management can be done to prevent obstipation. This treatment consists of removal of impacted feces, maintaining hydration, laxative therapy and prokinetics agents. Diets may be individualized for this problem and diet recommendations depend on how much motility is present in the large intestine. If there is some motility present, a diet such as Royal Canin Gastrointestinal High Fiber® is advised. If using other food, fiber supplementation with psyllium (1-4 tsp per meal) or pumpkin (1-4 tbsp. per meal) may help. Canned food can increase water intake and can have supplements added. If the patient starts impacting on the high fiber diet then try a high digestible diets with low residue such as Purina EN®, Hills ID® or homemade diets.

Lactulose (0.5 ml/kg q 8 – 12 hr.) is a good hyperosmotic laxative. Polyethylene 3350 Powder (Miralax®) has been also used in these cats (1/8 – 1/4 tsp BID with food).

There is no good evidence based in vivo studies to show that the prokinetic cisapride is effective, but anecdotally it probably helps with mild to moderate constipation in the cat.

Megacolon may not be medically controlled and surgical colectomy can be performed.

What tests do I submit for immune mediated disease?

Immune-mediated diseases include non-organ specific (systemic lupus erythematosus) to organ specific (immune mediated hemolytic anemia [IMHA]) disease. Inquiries about IMHA and immune mediated thrombocytopenia (IMT) are common.

Cases with IMHA are usually presented for weakness, lethargy and possibly icterus. General examination can show pale yellow mucous membranes, tachycardia and heart murmur. Basic laboratory work (CBC, serum biochemistries, urinalysis) submitted shows a regenerative anemia, polycythemia, spherocytosis, neutrophilia, increased liver enzymes and hyperbilirubinemia. In acute disease, reticulocytes may not be increased as it take 3 – 5 days for their appearance in the circulation. A cage-side slide agglutination test can confirm IMHA. A Coomb's test may not be necessary if the slide agglutination test is positive.

The etiology of IMHA can be infectious, neoplastic, drug/vaccine administration or primary.

These differential diagnoses must be ruled out prior to a diagnosis of primary IMHA.

Canine infectious agents that are known to cause IMHA include: rickettsial (*Ehrlichia canis*), protozoal (*Babesia* species) and bacterial (*Mycoplasma*, *Leptosporosis*). Therefore tests that are

routinely submitted include tick borne diseases (Rickettsial, Ehrlichia, and Babesia). We also start the patient on doxycycline on admission while awaiting results.

In middle-aged or older animals, imaging should be completed to investigate neoplasia. This imaging includes chest and abdominal radiographs as well as abdominal ultrasonography.

Immunology testing can include Coomb's test and anti-erythrocyte antibodies. ANA does not test for IMHA and should only be used if SLE is suspected due to multiple organ involvement.

IMT can also be caused by infectious, neoplastic, drug administration or primary. Infectious agents that can cause IMT include *Anaplasma phagocytophilum*, *Ehrlichia canis*, *Anaplasma platys*, *Babesia species*.

Immune mediated polyarthritis (IMP) is another disease seen in dogs that can be caused by infectious, neoplastic, vaccine/drug or be idiopathic. These dogs present with short-gaited strides, painful joints and possible fever. Infections with bacteria (*Borrelia*, *Mycoplasma*), rickettsia (*Ehrlichia ewingii*, *Anaplasma phagocytophilum*), protozoa (*Leishmania*) and viruses (Calicivirus in cats) can cause polyarthritis. Other antigen sources such as endocarditis, discospondylitis, pyelonephritis or urinary tract infection, periodontal disease, skin disease, gastrointestinal disease or heartworm disease must also be ruled out.

What is the best immunosuppressive for immune mediated diseases?

Due to the lack of evidence based studies in IMHA and ITP, there is no true recommendation for immunosuppression except for corticosteroids. Glucocorticoids are the mainstay of treatment. Dosage of prednisone (or prednisolone) is not standardized and ranges from 0.5 - 4 mg/kg/day. The usual initial dose is 1-2 mg/kg/day. There is a reported high dose pulsed approach but no clinical studies have been completed to assess effectiveness. In any immune mediated disease, the dose of glucocorticoids should not be tapered until normalization has occurred and then the tapering should be done slowly over weeks. If the tapering is too rapid, the disease may return and be more difficult to control.

The immunosuppression from glucocorticoids may not be sufficient in some immune mediated cases and a second drug can be considered. Azathioprine, cyclosporin and mycophenolate are the most common drugs chosen. The addition of these drugs may allow for increased immunosuppression and faster tapering of the glucocorticosteroid.

Azathioprine is a thiopurine which interferes with purine synthesis. DNA and RNA syntheses are inhibited. The effect is on cell mediated immunity and causes a reduction in lymphocyte numbers and T cell dependent antibody synthesis. There is a lead-in time for effect and may be up to 7 days or longer. The adverse effects recorded include: myelosuppression, acute pancreatitis, gastrointestinal problems and hepatopathy. There is a breed related variation in metabolism of the drug due to an enzyme (thiopurine methyltransferase) activity. Giant schnauzers may be more sensitive to the drug as this enzyme activity is lower than in other breeds.

Mycophenolate is another drug that blocks the purine synthesis. It was developed as an alternative for azathioprine. Therefore, do not use the two drugs simultaneously. It has a faster

onset of action and lower toxicity than azathioprine. However, there have been reports of gastrointestinal effects in dogs.

Cyclosporin reduces IL-2 production from T cells and thus reduces proliferation of T cells and consequently B cells. Most research has been done with immune mediated skin disease but cyclosporin is being used more commonly with other immune mediated diseases. The adverse reactions include gastrointestinal effects, possible renal toxicity and emergence of oral papillomatous lesions.

Vincristine is not utilized for immunosuppressive effects but is used for management of IMT. Vincristine stimulates megakaryocytes to increase release of platelets and impairs phagocytosis of opsonized platelets. The action is through binding to tubulin and disrupting mitosis. This drug is used as an initial therapy and not continuous.

Leflunomide is a drug that decreases pyrimidine production and inhibits T and B cell proliferation. It has been used in cases refractory to conventional drugs or when glucocorticoids are contraindicated or non-acceptable.

Human Intravenous gammaglobulins (IVIG) competitively inhibits the binding of IgG to monocytes due to saturation of Fc receptors. It has been used in dogs with IMHA and IMT with life threatening disease. A benefit was seen in one study with IMT but no controlled studies have been done with IMHA. This is an expensive treatment and may cause allergic reactions.

Remember that cases of IMHA are hypercoagulable and may benefit from aspirin or clopidogrel therapy.

Do cats get IMHA?

Yes. Remember that infectious causes such as *Mycoplasma* and *Cytauxzoonosis* must be ruled out or treated.

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COMMON QUESTIONS FROM RDVMS REGARDING THE ENDOCRINE SYSTEM IN DOGS AND CATS

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Why is this patient drinking so much??

Polyuria/polydipsia is a presenting complaint for many conditions and in fact one article in the literature lists 28 causes. When the mechanism of urine concentration is reviewed, then one can look at different areas that can affect that concentrating ability. The renal tubule concentrates using the concentration gradient that is developed and maintained by the loop of Henle with sodium and urea and also the presence and response to ADH. The tubule is also prone to osmotic diuresis which is seen when the proximal tubules are not able to resorb or have reached the maximal transport ability as seen with glucosuria. With the understanding of these mechanisms a system of looking at the causes can be used to develop a list of differential diagnosis.

An apparently healthy dog can present for PU/PD with no abnormal physical finding. History must be thorough to investigate diet, drugs and possible toxins. Also important information is spay status, other health problems or any environmental or behavioural changes.

Initial diagnostic approach includes a complete blood count, serum biochemistries with electrolytes and a urinalysis with culture. These simple tests can rule out many common diseases.

The urinalysis can confirm polyuria by examining the specific gravity. A urine with low specific gravity is difficult to evaluate for infection and therefore culture is required to rule out pyelonephritis. If the urine is concentrated, the complaint is not a polyuria but may be a pollakiuria and diagnostics for that problem can be pursued. Glucosuria may be an indication for diabetes mellitus or a tubulopathy such as primary renal glucosuria or Fanconi's syndrome. Chronic renal failure, liver disease, electrolyte imbalances (sodium, potassium) and hypercalcemia are hopefully identified on the basic blood work. Endocrine diseases may be investigated with further testing (thyroid, Cushing's, Addison's disease).

Other differential diagnoses include: partial ureteral obstruction, splenomegaly, intestinal leiomyosarcoma, polycythemia, pyometra, leptospirosis, primary aldosteronism, acromegaly, pheochromocytoma, post-obstructive diuresis and renal medullary solute washout.

Diabetes insipidus is also a cause but can be difficult to diagnosis. When other differential diagnoses are ruled out, the usual suspects are DI, primary psychogenic polydipsia or Cushing's. Water deprivation tests can be time consuming and for safety for the animal should only be done once major differential diagnoses have been ruled out. Prior to water deprivation testing, a trial with DDAVP can be done, but it must be remembered that other diseases can respond to the supplementation with ADH.

How do I diagnose Cushing's disease?

Cushing's disease in dogs may present with PU/PD, polyphagia, skin disease (alopecia), pot-bellied appearance, hepatomegaly, muscle weakness, or hypertension. Other less common signs include hyperpigmentation, thin skin, poor hair regrowth, insulin resistant DM, urine leakage and possible recurrent urinary tract infections. Initial blood work may reveal an increased serum

alkaline phosphatase (ALP), increased ALT, hypercholesterolemia, hypertriglyceridemia, neutrophilia, lymphopenia, eosinopenia, thrombocytosis and mild erythrocytosis.

Cushing's disease can result from either a tumor in the pituitary gland (Pituitary dependent hyperadrenocorticism – PDH) or the adrenal gland.

The screening tests for hyperadrenocorticism that are available include: ACTH stimulation, low dose dexamethasone suppression and urine cortisol/creatinine ratio (UCCR). However, no test has 100% diagnostic accuracy.

The UCCR ratio is used frequently in Europe and has not been embraced in North America.

Urine should be collected at home, in the morning and at least 2 days after a visit to the veterinary hospital. The urine can be pooled and brought in to be tested. Other diseases or stress can falsely elevate this test result.

The ACTH stimulation can be used to diagnosis Cushing's disease, hypoadrenocorticism and to confirm iatrogenic Cushing's disease. It can be done at any time of the day but the effect of feeding is unknown. Synthetic ACTH preparation should be used and compounded should be avoided. The dosage is 5 microgram/kg IV with blood samples drawn before and 60 minutes after administration. Post values about the laboratory cut-off range indicates

hyperadrenocorticism and further testing is necessary to differentiate PDH from adrenal origin. Low dose dexamethasone suppression is done using 0.01-0.015 mg/kg dexamethasone sodium phosphate or polyethylene glycol IV. Blood samples are taken at time 0 (prior to administration) and then 4 hours and 8 hours after administration. Avoid feeding and try to decrease stress during the day (ie. No other diagnostic testing). Normal dogs suppress below the cut off values of the lab. If minimal suppression or an inverse (escape) pattern is seen, then further differentiating testing should be done.

Some cases can be very difficult to diagnosis because clinical signs indicate Cushing's disease but the screening tests do not support the diagnosis. These are referred to as "occult" cases. An ACTH stimulation test including sex hormones is offered at the endocrinology lab at the University of Tennessee. However, sex hormones have not been proven to cause "occult HAC". An indication to use this test is if there is inappropriately low cortisol concentration on the screening tests. Further investigation into the role of excess basal cortisol secretion is currently under way at the University of Tennessee.

Differentiating tests include: High dose dexamethasone suppression test, endogenous ACTH and abdominal ultrasound.

How do I treat Hyperadrenocorticism?

Once Cushing's disease is diagnosed and determined to be either PDH or adrenal origin, treatment options can be explored. PDH treatment is aimed at controlling the hyperadrenocorticism resulting from the pituitary tumor. Surgery or radiation of the primary tumor is not considered an option at this time in North America. To control the excess corticosteroid production, the medical options are mitotane, ketoconazole and trilostane. Medical treatment may be individualized as not all patients respond to each treatment. Trilostane is the only FDA approved drug for veterinary use. Initial dosage of trilostane is low (1-2 mg/kg q 12 hr) and can be monitored and adjusted.

If an adrenal tumor is found, surgery may be considered. Medical management is an option for stabilizing a patient. A CT scan should be done prior to surgery to determine the extent of

growth and invasion of the tumor into surrounding structures such as the caudal vena cava. Some tumors are not resectable.

What is a pheochromocytoma?

An adrenal mass can arise from the cortex (corticosteroid or aldosterone producing) or the medulla (catecholamine producing) of the adrenal. Pheochromocytoma is a tumor of the adrenal medulla that secretes catecholamine. The pheochromocytoma has become more recognized with the increasing use of ultrasound in veterinary medicine. Pheochromocytomas are slow growing and vascular. The size can vary and can grow large and invasive to the adjacent vessels (caudal vena cava).

This secretion of epinephrine is episodic and causes vague clinical signs. Weakness and collapse are key signs but panting, tremors, anxiety, PU/PD, gastrointestinal signs, and hemorrhage have been reported. Hypertension may be recorded on a physical examination but because of the episodic nature, obtaining a normal blood pressure does not rule out this condition.

There are no consistent physical examination or basic laboratory abnormalities for this disease. Initial diagnosis of an adrenal mass is made with ultrasonography as these masses are not usually palpable. Adrenal cortical masses are usually suspected due to the signs of hyperadrenocorticism and ultrasonography is part of the differentiating testing for this disease. However, abdominal ultrasonography may be completed for other medical reasons and an adrenal mass may be incidentally discovered.

To diagnosis pheochromocytoma, urine catecholamine concentrations can be measured. To differentiate between pheochromocytoma and hyperadrenocorticism, serum inhibin can be measured in neutered animals.

Treatment for pheochromocytoma is surgery. A CT scan is usually performed prior to surgery to evaluate the extent of vascular invasion. Medical management includes controlling blood pressure and arrhythmias.

When should I think about Addison's disease or hypoadrenocorticism?

Hypoadrenocorticism is the great pretender. It is not common in dogs but it is not rare.

Hypoadrenocorticism results from atrophy or destruction of the adrenal cortices and results in deficiency of both glucocorticoids and mineralocorticoids. Atypical hypoadrenocorticism is defined as having only recognized glucocorticoid deficiency. However these cases may have minimal mineralocorticoids and probably need supplementation.

Causes include primary (Addison's), idiopathic (which may be immune-mediated), iatrogenic, or destruction (metastatic neoplasia, granulomatous, or hemorrhage).

Most cases are young to middle aged dogs but the range reported is 2 months to 14 years.

Breeds that have a predilection include standard poodles, bearded collies, and leonbergers.

However many other breeds have been identified.

Clinical signs can be chronic waxing and waning or can be an acute crisis. Chronic signs can be vague and can include the gastrointestinal or renal systems. These signs may be brought on by

stressful situations. Knowing that many breeds can be affected and that age is not always typical, one should consider this disease in many cases that are having vague clinical signs. Acute cases can present with dehydration, hypotension, shock, painful abdomen, hypothermia, bradycardia with history of gastrointestinal signs. Those cases must be dealt with on the emergency basis with aggressive fluid therapy and correction of electrolyte and acid/base abnormalities.

Initial diagnosis is based on history, physical and clinical signs and basic blood work. A complete blood count (CBC) should be examined for a stress leukogram. Absence of lymphopenia or neutrophilia in a stressed, ill dog should be a flag. A normocytic, normochromic anemia may occur. Serum biochemistries can show an azotemia with an inappropriately concentrated urine leading to an incorrect diagnosis of primary renal failure. Hypoadrenocorticism is a cause of polyuria/polydipsia with a non-concentrated urine. In a stressful condition, the dog can become dehydrated causing increased BUN and creatinine which leads to the misdiagnosis of renal failure. The azotemia usually resolves quickly with fluid therapy. The electrolyte abnormalities are the usual flags for suspicion. The increased potassium and the decreased sodium with a sodium/potassium ratio of less than 27 is the classic presentation. However, in atypical cases, the electrolytes can be normal. It must also be remembered that there are other causes of increased potassium and decreased sodium. These include gastrointestinal disease, effusive disorders, renal disease and acid/base disorders.

Baseline serum cortisol concentration can be a screener for the suspicious cases. If the resting cortisol is above 2 microgram/dl the case is unlikely to have hypoadrenocorticism. However, definitive diagnosis requires an ACTH stimulation test. This test can be done even in the critical patient. If exogenous glucocorticoids are required in the crisis, dexamethasone can be used as it does not interfere with the cortisol assay. Other glucocorticoids (prednisone, hydrocortisone) will interfere with the test.

What is best way to measure blood pressure?

Blood pressure is being recognized as part of a complete physical examination in veterinary medicine. However not all veterinary clinics have the instruments to make this a reality. Direct blood pressure measurement is the gold standard but is not practical for practitioners. Indirect blood pressure measurement can be obtained using ultrasonic Doppler flow monitors or automated oscillometric devices. The oscillometric method uses an automated system with a cuff that records the oscillations of the vessel wall at different pressures and computes the systolic, diastolic, and mean arterial pressures. The Doppler flow method requires the use of an inflatable cuff attached to a sphygmomanometer placed on an appendage or tail and a piezoelectric crystal positioned over an artery distal to the cuff. Systolic (SAP) and diastolic (DAP) pressures can be obtained, though diastolic pressures readings can be inaccurate with Doppler flow method and require increased training to determine.

Size of the cuff used is important to obtain the most accurate measurement. The width should be approximately 40% the circumference of the appendage. If the width is too small, an increased pressure is measured and if too wide, then the pressure can be measured incorrectly low.

Positioning of the patient is a key factor for correct blood pressure reading. The cuff should be positioned at the level of the heart to most accurately reflect the arterial pressure. When using the Doppler flow method, the paw or tail may be clipped to allow a better contact with the crystal but should be done well in advance of the procedure to decrease stress. The patient should also be acclimated to the room for at least 5 – 10 minutes to decrease stress. In cats, the limbs may be used, but the tail may be a better option.

The first measurement should be discarded and then the average of 3 – 7 consistent measurements should be calculated. The appendage used, the cuff size and the readings should all be recorded in the medical record so consistency can be used for future measurement and comparison.

Different studies have reported normal pressures but most record a normal < 160 mm Hg. However, healthy sight hounds may have a resting systolic blood pressure that is greater than 15 mmHg higher than other breed.

Why do I care about hypertension?

Hypertension can result from “white-coat” stress and this should be determined by decreasing any possible stress for the patient during measurement and repeated measures over time.

Hypertension can be primary or secondary. In our veterinary patients, we recognize secondary hypertension more commonly and this is associated with renal disease, hyperadrenocorticism, diabetes mellitus, and hyperthyroidism. Other less common causes are pheochromocytoma, primary aldosteronism and acromegaly.

Hypertension may be a reason to test for the mentioned conditions but in itself it is damaging to the body. The target organs are the eyes, the kidneys, the brain and the cardiovascular system. Acute blindness in cats may result from retinal hemorrhage or detachments and may be the reason for presentation. Hypertension causes proteinuria which is very damaging to the tubule; however, renal disease can lead to hypertension.

Controlling documented hypertension along with the underlying disease can decrease further progression of complications of hypertension.

What’s the best test to diagnose a hyperthyroid cat?

Hyperthyroidism usually affects older cats but the age range is reported to be 4 – 22 years. Most cats present with clinical signs of PU/PD, polyphagia, weight loss, gastrointestinal signs and skin changes. Other clinical signs include: respiratory signs, weakness, tremors, decreased appetite, and decreased activity. General examination may reveal weight loss, a palpable thyroid gland, tachycardia or gallop rhythm, heart murmur, skin changes and hypertension.

Since other diseases are considered with many of these clinical signs, a complete blood count, serum biochemistries and urinalysis should be completed.

Serum biochemistries may show increased liver enzymes (ALT, ALP) and increased blood glucose. The urine is usually concentrated unless renal insufficiency is also present. The hyperthyroidism may mask underlying kidney disease due to the increased GFR.

The screening test for hyperthyroidism is a total T4 (TT4) concentration. The RIA method is the gold standard for measuring TT4. There are other methods for measuring that are employed by different laboratories (chemiluminescent immunoassay, ELISA, enzyme immunoassay method – EIA).

Free T4 (fT4) should not be used as a screening test as the specificity is poor (false positives). Be cautious of the method of measuring fT4 as not all laboratories use the same methods (equilibrium dialysis, RIA, chemiluminescent immunoassay). fT4 should be used in conjunction with TT4 and clinical signs.

Serum T3 concentrations are not useful for diagnosis because in one study by Peterson et al, one-third of the hyperthyroid cats had normal T3 concentrations.

TSH measurement is also a test that should not be used routinely to diagnosis hyperthyroidism in cats as results can be over interpreted. If used, it should be interpreted with TT4.

Thyroid scintigraphy uses nuclear medicine to display functional thyroid tissue. This can be used in suspected cases that have normal or borderline TT4. It could be considered the gold standard; however availability is the challenge.

There are cases in practice that have all the clinical signs of hyperthyroidism, have other differential diagnoses excluded but do not have abnormal TT4. If scintigraphy is not an option, then a T3 suppression test can be considered. This test measures serum T3 and TT4 concentrations prior to testing. T3 (liothyronine) is given orally (20 micrograms q 8 hr for 7 doses). A blood sample is then taken 2 – 4 hours after the last dose and the TT4 and T3 serum concentrations are measured. The T3 concentration confirms that the cat received the drug. The T3 should suppress TT4 in normal cats. Therefore if TT4 is >20 nmol/l +/- $<35\%$ suppressed from baseline, hyperthyroidism is diagnosed.

What is the best treatment for a hyperthyroid cat?

Hyperthyroidism can be treated either medically, surgically or with radioactive iodine. Medical management with methimazole can be challenging in that the cat must be medicated once to twice a day for the rest of its life. Methimazole can be administered as an oral tablet or liquid or as a transdermal form. The transdermal may have less gastrointestinal side effects than the oral form. However, it may be necessary to increase the dose with long term treatment. Medical management requires routine blood monitoring to insure no biochemical abnormalities and adequate treatment.

Medical management may also unmask underlying renal insufficiency. The return to normal GFR may reveal an azotemia and therefore these cases must be monitored closely for renal failure. Management of both diseases is then undertaken.

Some cats cannot tolerate methimazole and will have clinical side effects such as gastrointestinal upset, facial pruritus or biochemical side effects such as increased liver enzyme concentrations, thrombocytopenia, neutropenia or anemia. These cases may not be able to tolerate medical management.

Surgical treatment is an option if the thyroid gland or glands can be palpated. The glands may not be palpable if they have moved into the thoracic inlet. The surgical cases may be managed medically initially to evaluate renal function as well as to stabilize weight and heart issues. Anesthesia is a concern as well as postoperative monitoring. These cases require intensive care post surgically to monitor for hypocalcemia as the parathyroid glands can be damaged during surgery as well as the recurrent laryngeal nerve.

Radioactive iodine is a non-invasive method of treatment but requires authorized facilities. These cases must also be stable for this treatment. Medical management with methimazole is suggested to unmask renal disease and to stabilize the patient. The cat is treated for 14 days and then the serum TT4 is measured. If the TT4 concentration is in the normal range, a serum biochemistry is done to evaluate the renal function. If the renal function is normal, then the methimazole is discontinued for 2 weeks prior to radioactive iodine treatment. This allows uptake of the iodine by the abnormal thyroid tissue. The disadvantage of radioactive iodine is that the cat is in radiation isolation for many days depending on the State. These cats cannot be handled, evaluated or treated with other medication during this time. This fact adds to the decision of treating cats with predisposing illnesses.

A second radioactive iodine treatment may be required in a low percentage of cats (2-5%). However, radioactive iodine treatment is less stressful, does not require anesthesia and will end medical treatment.

Why is this Diabetic not controlled?

A diabetic pet may require increased insulin doses to remain regulated or may become unregulated while on insulin therapy. Insulin resistance is suspected if the insulin dose is greater than 2.2 U/kg to maintain control. Loss of control and persistent marked hyperglycemia can occur and doses of 1.5 U/kg may not have any effect on the blood glucose measurement.

The clinical signs of uncontrolled DM can include polyuria, polydipsia, polyphagia, weight loss or a more severe presentation of diabetic ketoacidosis. The approach to the patient depends upon the presentation. All cases are evaluated with a physical examination and basic blood work and urinalysis. The less critical cases may also be evaluated with a blood glucose curve and/or serum fructosamine concentration. If the blood glucose curve or serum fructosamine indicate non-regulation, then the investigation begins.

The first questions to ask are in regards to the owner and insulin. The age, the care, handling, and administration of the insulin are important. Has there been a change in caretaker of the pet? Is the pet on other medications? Is the pet neutered? These are easy questions to rule out simple answers to the problem.

The next question is whether or not this animal is being overdosed with insulin and is experiencing the Somogyi effect. This can occur when the insulin dose is slowly increased without regular blood glucose measurements. The Somogyi effect can occur soon after insulin administration and must be measured at that time, as the blood glucose level will rapidly increase due to the counter-regulatory hormone secretion and. If the blood glucose is only spot checked throughout the day, it would appear that the blood glucose remains high. If the animal is experiencing this Somogyi effect, the simple answer is to lower the insulin dose back to the initial dose and start the regulation again.

If the Somogyi is not occurring and the blood glucose is truly increased throughout the day, then causes of insulin resistance must be explored. The most common causes of resistance include endocrine diseases and infections. In dogs, these causes include hyperadrenocorticism, bacterial infections, hypothyroidism and diestrus. In cats, the causes include hyperthyroidism, bacterial

infections, and organ insufficiency (renal, liver, cardiac), and other less common causes (acromegaly, hyperadrenocorticism).

Infections cause secretion of the counter regulatory hormones (glucagon, cortisol, and epinephrine). Diabetics are prone to urinary tract infections due to the low urine concentration and the glucosuria (great media for bacterial growth). Evaluation of just a urinalysis for infection is not adequate for diabetics. A bacterial culture must be submitted to rule out an infection. Other sites of infection/inflammation such as periodontal disease and skin infections must be ruled out.

Endocrine diseases cause insulin resistance. Hyperadrenocorticism is most common in dogs and hyperthyroidism in cats. The challenge of identifying concurrent hyperadrenocorticism and diabetes can be challenging. An uncontrolled DM, may have a falsely increased cortisol with either ACTH stimulation test or LDDS test. If possible, stabilizing a DM with increased insulin dose can help with interpretation of these tests. However, once diagnosed with hyperadrenocorticism, treatment for that disease will drastically decrease the insulin requirements and close monitoring must be done to avoid insulin overdosing.

Other causes of insulin resistance include obesity, acromegaly, hypothyroidism, chronic pancreatitis, pheochromocytoma, other cancers, hyperlipidemia and renal insufficiency.

The production of insulin antibodies can occur but is not the usual cause of insulin resistance. Therefore, all other causes must be ruled out prior to diagnose.

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COMMON QUESTIONS FROM RDVMS REGARDING THE URINARY SYSTEM IN DOGS AND CATS

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How do I treat canine urinary incontinence not responsive to standard therapy?

Standard initial treatment for urinary incontinence in spayed female dogs is with reproductive hormones (e.g. estriol, premarin or diethylstilbesterol) or sympathomimetic drugs (phenylephrine, pseudoephedrine and phenylpropanolamine). Combining these two classes of drugs can be attempted to achieve continence if single treatment is not effective. Further diagnostic tests should be explored for dogs that are not responding to pharmacological treatment. Basic urinalysis plus culture should always be completed to evaluate for concentrating ability and possible infections. A patient with polyuria/polydipsia may be misdiagnosed as primary incontinence. Additionally, CBC and serum biochemistries should also be performed. Imaging of the bladder (radiology and ultrasonography) can determine if anatomical abnormalities may be contributing. In select cases, contrast urethral radiology can determine intra or extra-luminal problems. Urethral pressure profile (UPP) is useful to determine the pressures at different locations in the urethra. However, this test is only done at select locations. Urethral scoping can visualize the urethral and bladder mucosa and can determine if ectopic ureters are present. If there is no underlying problem found, urethral bulking or surgical placement of a percutaneously adjustable hydraulic urethral occluder may be considered as treatment options.

When should I worry about urine crystals in an asymptomatic patient?

Urinary crystals can indicate an underlying problem or may be insignificant. In the bladder, crystals are formed in supersaturated urine. Once urine is collected, crystals can form or dissolve with changes in pH, temperature, or technique of preparation. Importantly these changes can alter interpretation of results. Evaluation of recently acquired urine samples is preferred to determine crystal significance. Crystals should be interpreted in view of the entire urinalysis in relation to pH and specific gravity. Crystalluria may indicate a potential risk for urolith formation. While crystalluria does not predict urolith formation, there is an increased risk if heavy crystalluria in a fresh urine sample is present. If this occurs, dietary changes are not always indicated; however, increased water intake and increased surveillance are warranted. Early detection of uroliths may aid in removal by noninvasive means if the uroliths are small.

Magnesium ammonium phosphate (struvite) crystals can be found in urinalysis of normal dogs and cats. These crystals also can occur with urinary bacterial infection due to urease-induced production of ammonia. Therefore the finding of these crystals in dogs or especially in pediatric dogs and cats in addition to finding pyuria or bacteriuria in the urinalysis should prompt automatic culture of the urine sample. Urolithiasis should be investigated if there is an infection plus crystals noted on the urinalysis. However, mature cats with struvite crystalluria usually do not have an underlying infection. Feline lower urinary tract disease (FLUTD) or feline

interstitial cystitis (FIC) sometimes presents with excessive struvite crystalluria and struvite crystals remain the most common mineral component of urethral plugs.

Calcium oxalate crystalluria can be a normal finding but if significant quantities are present then diseases such as hypercalcemia or ethylene glycol toxicity should be explored. Interestingly, patients with calcium oxalate urolithiasis only have crystalluria 50% of the time. To decrease risk of stone formation in cases of persistent crystalluria, medical management with diet and oral potassium citrate may be necessary.

Normal dogs and cats do not usually have urate crystals but they can occur. Dalmatians and English Bulldogs are breeds that are prone to this type of crystal and therefore are at risk for urolithiasis. In other breeds and cats, if urate crystals are present the animal should be evaluated for underlying liver disease such as portal vascular anomalies (shunts).

When to worry about proteinuria in an asymptomatic dog or cat?

Proteinuria discovered on a urine dipstick or by screening microalbuminuria test may be an incidental finding but should be further investigated. Presence of protein should be confirmed with SSA (sulfosalicylic acid); however, SSA may still yield inaccurate results. The source of the protein must be investigated. Positive results occur with hemoglobinuria and myoglobinuria. For a true proteinuria due to glomerular disease, urine sediment must not contain cells or bacteria. The first step is to microscopically evaluate urine sediment and to perform a urine culture. If there is inactive urine sediment and culture is negative, then a urine protein/creatinine ratio (UPC) should be performed to verify and quantitate the proteinuria. To verify true persistent proteinuria, serial UPC measurements are recommended as daily variations can occur. UPC is considered abnormal at >0.5 in a dog and >0.4 in a cat; however, most healthy dogs and cats have $UPC < 0.2$.

When post-renal proteinuria is excluded, confirmed proteinuria is usually an indication of glomerular protein loss. The exception is seen with some tubular disorders such as Fanconi's syndrome. Glomerular disorders can be either primary or secondary in origin and proteinuria should be investigated. Secondary causes of glomerular disease can be due to antigenic reactions; therefore a search for underlying antigenic stimulus such as non-renal infection or heartworm disease must be made. Hypertension can damage the glomerulus leading to proteinuria and therefore systemic blood pressure measurement is indicated. Cushing's disease or oral corticosteroid therapy in dogs will cause a mild to moderate proteinuria that can resolve with treatment or cessation of therapy.

If an underlying cause cannot be found, renal biopsy should be considered. Since glomerulonephritis has different histopathological forms, analysis will help with descriptive analysis as well as determination of future therapy.

How to deal with nephroliths?

The best treatment plan for a nephrolith depends on the type, the impact on the patient, and the potential morbidity of available treatment options.

Nephroliths can vary in size from small to staghorn and must be differentiated from renal mineralization. Nephroliths may be part of the primary problem or may be an incidental finding on survey radiographs. Mineral composition may be difficult to determine without removal and analysis. In cases where this is not possible, the most likely composition can be inferred from radiographic appearance, urinalysis (crystalluria and pH), presence of infections and evaluation of serum mineral levels (calcium).

Calcium oxalate are the most common nephroliths in cats. Surgical removal can cause renal damage and therefore feline calcium oxalate nephroliths are best managed medically; however, with proximal ureteral obstruction, surgery may be necessary. As dissolution is not possible, the goal is to prevent growth of the nephrolith. Use of lithotripsy (extracorporeal) is not indicated because the feline kidney is unlikely to tolerate the shock wave dosage required for complete fragmentation and ureteral passage of stone material. Medical management of cats includes evaluation for hypercalcemia or chronic kidney disease. Mild hypercalcemia, azotemia and calcium oxalate nephrolithiasis commonly co-present in feline patients. Therapy for idiopathic hypercalcemia is aimed at decreasing serum calcium concentration which includes diet, fiber and possibly bisphosphonates. Nephroliths in a solitary functioning kidney (big kidney-little kidney syndrome) may pose additional concern but still are usually best managed with medical strategies.

Many dog breeds seem predisposed to calcium oxalate nephroliths. However, suspecting this type of nephrolith in dogs should also lead to investigation of underlying disease such as hypercalcemia or endocrinopathy (Cushing's). Treatment is similar to cats with the exception that extracorporeal lithotripsy may be used if conditions are suitable (size of nephrolith and patient stability). Surgical removal also may be indicated when a nephrolith is associated with pain, infection, or obstruction and renal pelvic dilation.

If struvite nephroliths are suspected they can be treated with dissolution diets and antimicrobial therapy (if infection-induced). Ureteral obstruction is a possibility as the nephrolith becomes small enough to drop into the ureter; however, this appears rare in actual practice.

Are there any new treatments for feline lower urinary tract disease?

Many differential diagnoses show similar clinical signs for feline lower urinary tract disease (FLUTD). It is important to rule out these diseases prior to making a diagnosis of feline idiopathic cystitis (FIC). Diagnostic tests including urinalysis, urine culture, basic blood work (CBC, serum biochemistries) and imaging should be completed in all cases, with prioritization of the urinalysis and survey radiographs. A presumptive diagnosis of FIC can be made if no underlying disease is found. Triggers for FIC episodes are not always known and could include stress or neurogenic stimuli. Therefore multi-modal management to prevent these recurrences includes stress reduction (environmental enrichment, facial pheromones) and more traditional therapies of diet (moist food), increasing water intake and analgesics.

Chronic cases of FIC include those that continue to have clinical signs without remittance or that have frequent recurrences even after multi-modal management. These cases should be provided appropriate pain management to break the pain cycle using short acting analgesics such as buprenorphine, butorphanol or NSAIDS (meloxicam, robenacoxib). Amitriptyline treatment may

reduce recurrence by decreasing stress or via other pharmacologic effects. Feline facial pheromones may be useful by decreasing stress. Treatment with the oral glycosaminoglycan, pentosan polysulfate (Elmiron®), given long term (months to years) may reduce clinical signs in some cats, despite a lack of strong evidence based research. If successful, it is a lifelong therapy. Other glycosaminoglycan drug usage in these FIC patients is also antidotal and future studies are necessary. Each cat therapy must be individualized to find the best management.

When to do a perineal urethrostomy on a cat?

Urethral blockage with mucous or mucocrystalline plug in a male cat can be very frustrating in both initial and long term management. When the initial blockage is difficult to resolve, urethral spasm can make recovery of voiding function difficult. The irritated urethra can spasm at any location and can involve smooth or striated musculature or both. Because of these spasms, prolonged recovery can involve multiple urinary catheter placements and therefore pharmacological therapy can help decrease the urethral spasming.

After this initial blockage, management with diet, increased moisture intake, and multi-modal environmental management may help prevent recurrence of urethral plugs. If several recurrences occur despite appropriate management, a perineal urethrostomy may be considered. For example, I will be more likely to recommend a PU be performed after a cat's third blockage. In other situations, a PU may be indicated as a salvage procedure when severe urethral trauma has occurred, or to alleviate blockage by a urethral stricture.

Perineal urethrostomy surgery is not innocuous. With loss of the distal urethra, normal barriers are lost; bacterial infection can occur and may ascend into the kidneys. Incidence of bacterial urinary tract infections increases with PU surgery and has been reported to occur in up to 50% of patients depending on the technique employed. PU surgery is delicate and if not done correctly, urethral leakage or stricture can occur. Finally, the cat may still suffer from episodes of non-obstructive lower urinary tract signs if medical management is not applied and continued.

Why are urine cultures always negative when I send them to the diagnostic laboratory?

Many practitioners do not submit urine for culture because it is expensive and they receive "false negatives" even when urinary tract infection is suspected. True and false negatives can be related to procedural and biological reasons.

Negative urine cultures are often accurate and reflect a sterile sample. In house use of urine dipsticks may give false information on the presence of leukocyte in dog or cat urine. Microscopic urine sediment examination may falsely overestimate "bacteria" as many artifacts mimic bacteria and even bacterial motion. The best sediment examination is achieved by using routine stains (clean Gram stain or new methylene blue) and qualified personnel.

Timing is important. Whenever possible, samples should be collected prior to antimicrobial treatment in new cases. If this is not possible then the diagnostic laboratory should be notified of current treatments. Antimicrobial administration may inhibit bacterial growth in culture but may not be bactericidal in vivo. Additionally, the laboratory must be notified if anaerobic and

mycoplasma organisms are suspected as these organisms will not grow with standard aerobic methods.

Adverse effects may occur with handling of the urine sample prior to transport. Samples should be refrigerated and transported as soon as possible. Do not ship formalin biopsy samples with urine samples for culture. During transportation, temperature may affect culture outcome. Long delays or high temperatures may affect the viability of bacteria.

Finally, it must be remembered that not all lower urinary tract signs are caused by bacterial infection. Other etiologies such as urolithiasis, neoplasia and idiopathic cystitis should be considered and investigated in patients with persistent signs and repeated negative culture results.

How to treat those urinary tract infections that keep coming back?

Repeated urinary tract infections can be classified based on the species and pattern of bacteria observed. Sequential urine cultures are invaluable in sorting out these bacterial urinary tract infections. Recurrent signs caused by the same organism are usually due to a relapse of the original infection. Signs due to a new reinfection are usually caused by a different organism. However, repeated *Escherichia coli* infections can be challenging to interpret as there are different strains of the bacteria. *E. coli* is a common urinary tract infectious agent as well as a common persistent organism in some patients.

“Recurrent” infection must be evaluated in relation to previous treatment. In a relapse (same bacteria), the effectiveness of the prior treatment plan is questioned: Was adequate treatment given to the patient (appropriate drug, dosage and duration)? Was urinalysis and culture rechecked after treatment completion to ensure clearance of infection? Was this a simple or complicated infection (especially pyelonephritis, prostatitis or UTI with concurrent disorders)?

With reinfections (different organisms), underlying predisposing causes must be investigated. These include problems with host defense such as anatomical abnormalities of the lower urinary tract (vaginal urine pooling, recessed vulva, or masses), urethral incompetence, neurological impairment of micturition, and alteration in urine volume and concentration. Uroliths can also act as a nidus for relapse or reinfection at any location in the urinary system. Other underlying systemic predispositions include immunocompromise such as endocrine diseases (hyperadrenocorticism or diabetes mellitus) or corticosteroid therapy. Unfortunately, an underlying disorder may not be detected in many cases.

Management of recurrent urinary tract infections can still be challenging even when a distinct reason or underlying cause can be identified. Surgical problems can be corrected and manageable systemic disorders can be addressed. Other predisposing disorders may or may not be able to be eliminated (e.g. poorly controlled endocrine disorders, irreversible neurological impairment). These cases should be managed as a complicated UTI with long term appropriate antimicrobial therapy. Pulse or low dose daily antibiotic therapy or other prophylaxis can be considered once the initial infection is cleared.

Many of these recurrent infections involve *E. coli*. These bacteria must first adhere to the cell and may actually be internalized. Oral therapies that may be used to prevent adhesion include cranberry extract and D-mannose. Cranberry extract contains proanthocyanidins that inhibit *E. coli* adhesion to epithelial cells. D-Mannose may also be an adhesion deterrent, but clinical studies of its efficacy are lacking.

How do I treat those highly resistant urinary tract infections?

Patients with unidentified or unmanageable underlying disease can develop resistant UTI. These cases have had many different classes of antimicrobials prescribed and as a result the bacteria have become resistant. As with recurrent UTIs, an underlying disease or condition should be investigated and treated if possible.

The diagnostic laboratory can be of great help in maximizing effective antimicrobial choices. Antibiotic susceptibility is reported via agar disc diffusion (Kirby Bauer) or by antimicrobial dilution technique (MIC – minimum bacteriostatic bacterial concentration). With MIC, each antibiotic is evaluated in respect to serum levels; however, urinary concentrations may be significantly higher. Therefore renally excreted antimicrobials may be effective even if the lab result reports intermediate susceptibility or resistance. Short term use of injectable antimicrobials may be necessary to eliminate the infection. If aminoglycosides are used, urinalysis should be monitored frequently for early renal tubular damage by using cast identification. If susceptible, infections can be treated with the antiseptic therapy nitrofurantoin but potential side-effects must be recognized. As discussed in the previous section, oral cranberry extract and D- mannose may be useful in management of some *E. coli* infections.

How about those asymptomatic (silent) urinary tract infections?

A patient may not show clinical signs of lower urinary tract disease even with a positive bacterial culture. Urine contamination must be ruled out first and bacterial numbers should be evaluated. Significant infection is likely if one or two high growth organisms are isolated from an appropriately handled cystocentesis urine sample. Patients with silent infections usually have an uncontrolled underlying disorder. In the course of frequent, deliberate urine cultures as part of a monitoring plan, infection may be discovered.

Initially, standard treatment approaches for complicated infections are pursued. These cases can be very frustrating because the infections may not be cleared and resistance may occur. These patients still do not show clinical signs but the concern is for ascending infection or if bacteria are urease producers for possible formation of struvite uroliths. In some cases, no treatment is successful in eliminating the organism. Inhibitory antimicrobial treatment (low dose, once daily administration) has been recommended to prevent complications. In other cases, all antimicrobial therapy may be discontinued and hopefully with time, resistant bacteria may be replaced with other susceptible bacteria. The urine should be re-evaluated and cultured after 1 to 2 months with hopes of finding a treatable infection. In other cases, treatment is withheld unless clinical signs of infection or urosepsis occur.

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COMMON QUESTIONS FROM RDVMS REGARDING THE RESPIRATORY SYSTEM IN DOGS AND CATS

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When faced with a kennel cough outbreak, what is the best method for testing?

Kennel cough is the old term for what is now called canine infectious respiratory disease (CIRD). The disease is commonly seen with kenneled dogs and is transmitted by direct or indirect methods. The list of infectious agents that have been recognized to contribute to this disease is growing. The most common bacteria identified are *Bordetella bronchiseptica* and *Mycoplasma*. *Streptococcus zooepidemicus* has been identified as a cause of serious pneumonia in some cases of CIRD. These bacteria can be cultured from a transtracheal wash (TTW) but special requests must be made for mycoplasma culture. The well known viruses involved include: parainfluenza, adenovirus-2, and respiratory coronavirus, and canine influenza virus. Other viruses identified include: canine hepatitis virus, bocavirus, pneumovirus, and pantropic canine coronavirus. Distemper virus can be mistaken for CIRD. These CIRD cases must be treated as infectious and if possible on an outpatient basis. If hospitalized they must be isolated.

For individual dogs, testing is determined by the seriousness of the condition. Most cases are uncomplicated with no fever, anorexia or lethargy. These cases are sent home and treated with antibiotics and antitussives. However, cases can become complicated if immunocompromised or have overwhelming infectious exposure. Those cases have a productive cough, naso-ocular discharge and fever. An interstitial and/or bronchopneumonia is identified on thoracic radiographs and blood work reflects the infectious/inflammatory nature. These serious cases are first radiographed and then washed to obtain samples for cytology and bacterial culture. For viral diagnostics, serology is used for canine influenza, urine PCR is used for distemper and PCR or virus isolation can be used for other viruses.

In a kennel situation, an outbreak of CIRD can be devastating. Most dogs are noncomplicated and recover with minimal to no treatment. However, since the transmission is through direct or indirect contact, many dogs become infected. To identify the pathogenic organism, affected dogs can be tested. Influenza may not be present at the time of clinical signs and paired serology may be needed to positively confirm diagnosis. Nasal swabs for virus and bacteria can be done but remember that some bacteria are part of the normal nasal flora. Finding a positive diagnosis does not always implicate the causative agent.

Management of a kennel outbreak is aimed at environment and isolation control. To decrease transmission, new dogs should not be admitted to the kennel. Affected dogs should be isolated from non-infected dogs and strict isolation standards followed.

What to do about chronic nasal discharge in a dog?

Chronic nasal discharge in dogs can be frustrating. History and signalment can aid in the approach. Unilateral discharge may lead one to consider a foreign body or a tumor but this is not always the case. Bilateral discharge can occur from infectious, inflammatory or neoplastic

causes. Epistaxis can be a local problem or indicate systemic disease (hypertension, thrombocytopenia and vasculitis).

Initial diagnostics depend on history and signalment and thus may involve basic blood work, urinalysis and thoracic imaging. More specific diagnostics involve nasal CT, rhinoscopy and biopsy. Skull radiographs do not provide information to the same degree as CT. CT allows evaluation of the nasal cavities, sinuses and also the extent of the disease. It can guide post-imaging rhinoscopy, biopsy procedures and also plan for future radiation therapy.

Nasal aspergillosis is the most common fungal infection in the nasal passages and is usually seen in young to middle aged dolichocephalic dogs. The fungal hyphae are confined to the surface of the mucosa and the body's inflammatory response results in bony destruction and turbinate loss. Treatment consists of local debridement and topical antifungal infusion. The frontal sinuses may be affected and must be included with treatment. Oral antifungal therapy is much less effective but may be used if the cribiform plate has been penetrated.

Idiopathic lymphoplasmacytic rhinitis is common but an underlying etiology is seldom discovered. This inflammatory response is likely an aberrant immune response to multiple antigenic factors. Other causes of nasal discharge must be ruled out prior to diagnosing this condition. Treatment is frustrating. Allergen avoidance is not usually helpful unless blatant exposure is known (secondhand smoke). Corticosteroids do not seem to help. NSAID therapy combined with antibiotics may help (piroxicam and doxycycline for 4 – 8 weeks if possible). Oral itraconazole can also be tried as hypersensitivity to commensal fungal organisms may be contributing to the condition.

Nasal neoplasia occurs in middle aged to older dogs. The majority are malignant and arise from within the nasal cavity. These are aggressive and cause destruction of nasal turbinates and surrounding bone. Metastasis occurs late in the course of the disease. Radiation treatment is the treatment of choice.

Anything new with upper respiratory disease in cats?

Upper respiratory disease in cats is one of the most frustrating conditions for a veterinarian. The most common cause is viral disease (herpes or calicivirus) with secondary bacterial infection. However, one must not ignore other differential diagnoses. Once these are ruled out with serology, nasal CT, rhinoscopy and biopsy, the usual disappointing result is probable viral disease with secondary bacterial infection. If the cat was infected as a kitten with virus, the nasal turbinates may have been infected with resulting loss of normal anatomy and defense methods. This will lead to a chronic discharge from secondary bacterial infection. The virus may not be able to be isolated in these cases in the chronic state.

How to treat these chronic cases? These cats can have bacterial infection resulting in copious nasal discharge. Submitting for bacterial culture of either discharge or nasal swab is not useful as the infections are opportunistic and will change with time. Periodic flushing of the nasal cavities under general anesthesia may relieve the buildup of discharge and this material is a better representation to culture. Be sure to use an endotracheal tube and pack off the nasopharyngeal area to avoid aspiration.

Broad spectrum antibiotics are indicated and should be based on the bacterial culture. However, the numerous times of flushing with anesthesia to obtain a culture is not practical and trial broad spectrum antibiotics can be used for 6-8 weeks. Repeat antibiotic use usually leads to resistant bacteria and *Pseudomonas* usually appears.

Antiviral drugs such as famciclovir will not work if the virus is not present. If the infection is recurring and is caused by herpesvirus, the drug may be a benefit. Oral lysine has been used but may not always be beneficial. This drug is also only indicated for herpesvirus infections.

Decongestants can improve mucosal edema through vasoconstriction but the use leads to a rebound effect. Antihistamines may dry out the nasal passage and this may lead to further inspissated material; however, some cats may respond. Other recently published research on intranasal vaccination to aid immunological response needs further work to advocate its use.

What about fungal pneumonia?

Fungal pneumonia is not rare in our region. Blastomycosis is the usual suspect. The cases are usually young male, larger breed dogs. However, we have seen cases in older, small breed dogs and even in indoor cats. The fungus lives in the soil and can be brought into the house on shoes or in house plants. The most sensitive and specific test is the urine antigen which is done by Miravista® laboratories. This measures the level of antigen and can be used for monitoring treatment. There is a new blastomycosis antibody test that when used in conjunction with antigen levels can aid in diagnosis.

Treatment is usually with itraconazole or fluconazole. Do not use compounded itraconazole. Our studies in both cats and dogs have shown that there is no absorption of compounded itraconazole and therefore the drug does not achieve therapeutic blood levels. In severe cases of blastomycosis or those with neurologic involvement, amphotericin can be used as a fungicidal treatment.

Anything new on feline asthma/bronchitis?

Research into feline asthma is continuing. The immune system's response to antigenic stimulation causes the hyper-reactivity and the inflammation associated with the condition. Treatment including antigen avoidance, anti-inflammatory corticosteroids and bronchodilators is still the main stay of therapy. Decreased exposure to trigger antigens would be ideal but not easily done. Changing cat litter from clay to paper will eliminate dust. Second hand smoke can also be a trigger as can perfumes, cleaning solutions or air fresheners.

Corticosteroids are the main anti-inflammatory drugs used for this condition but do have side effects. Oral prednisolone or prednisone are the usual initial systemic drugs chosen. Long acting injectable methylprednisolone acetate is not ideal and should be limited to those cats that are intractable or cannot be pill. The use of inhaled corticosteroid (fluticasone) is a more targeted treatment but requires a cooperative cat and owner. When starting inhalation therapy, there must be overlapped with oral corticosteroids for 7 – 10 days. Anti-inflammatory treatment is important because it has been shown that even while on corticosteroids, subclinical inflammation is still present in the lungs.

Bronchodilators assist with the bronchoconstriction that occurs with asthma and bronchitis. Oral therapy with theophylline can be used. Continuous use of albuterol inhalers has been shown to be detrimental in humans and cats as the drug can cause increased inflammation. Therefore inhaled albuterol should be reserved only as a rescue emergency therapy only. Another rescue therapy that owners can administer at home is subcutaneous terbutaline.

Other therapies that have been used in humans do not always translate to feline medicine. Antihistamines (cetirizine) and antileukotrienes (zafirlukast) have not been shown to help. Cyprohepatidine may be an adjunct therapy but is not as promising as once thought. Cyclosporin can be considered for use in asthmatic cats that may have conditions that contraindicate glucocorticoid therapy. The dose has not been determined but most use the same dose as that recommended for allergic dermatitis. Inhaled nebulized lidocaine has been used in human medicine and has been looked at in experimental cats. This may be another therapy that could be used as an adjunct to other treatments.

Skin testing for antigens is challenging because of the antigenic range that is possible. There have not been any studies to investigate the correlation between dermal reactions and inhalant antigens in clinical cats. Omega-3 fatty acids and luteolin supplementation may have some beneficial effects on airway responsiveness as shown in cats with experimentally induced asthma. This may also suggest that a diet change could be also tried.

How to treat a chronic cough in a dog?

Chronic cough in a dog can indicate inflammation, infection or neoplasia. Radiographs are always indicated. The next step in diagnostics depends on the results of the radiographs. If chronic bronchitis is suspected, a TTW is indicated for cytology and culture. Parasites can be the cause and should not be overlooked as they can be treated.

Collapsing trachea can be missed on radiographs even with inspiratory and expiratory exposures. Dynamic testing with fluoroscopy or endoscopy are used for diagnosis and to document the extent of the collapse. Initial treatment for collapsing trachea is usually weight loss and use of a harness. Medical management with antitussives and antibiotics (in case of secondary infections) can also be used initially. Short term anti-inflammatory doses of glucocorticoids can help with stabilization. Cerenia has been suggested for use as an antitussive for collapsing trachea but no evidence based studies support this treatment.

Severe respiratory distress from tracheal collapse can be alleviated with sedation. Acepromazine is useful for this scenario. The last resort treatment is tracheal stenting. This provides structure to the trachea but does not alleviate chronic cough nor future collapse distal to the stent.

Another cause of chronic cough in dogs is chronic bronchitis. This is usually seen in middle aged to older dogs. Diagnosis is by exclusion of airway infection, airway collapse or foreign body. Thoracic radiographs are important and may show increased bronchial to interstitial patterns. However, chronic bronchitis may not be as evident on the radiographs. Bronchoscopy can reveal tracheal or bronchial collapse, as well as airway hyperemia or abnormal mucosa. Airway samples are important for cytology and culture. Chronic bronchitis has cytological abnormalities of inflammation (neutrophils, eosinophils or mixed) and increased mucus.

Anti-inflammatory is the mainstay of therapy for chronic bronchitis. If bacterial culture has not been done, a doxycycline trial should be done prior to corticosteroid therapy. This is a long term therapy with corticosteroids, so a tapering regime should be used. Inhaled corticosteroids can also be used although systemic absorption does occur. Bronchodilators can help clinically with these patients although there is no effect of these drugs on the large airways. Cough suppressants should not be used if the cough is productive as this is the main method clearing secretions from the airway. The use of these drugs should only be used when inflammation has cleared or if there is tracheal/bronchial collapse. Weight loss can also help with this condition if the dog is overweight.

Older, small breed dogs can also have a chronic cough from heart disease. The left atrium can cause compression of the bronchi leading to irritation and cough. These cases must be managed for the heart condition and the cough can be controlled with an antitussive.

References available upon request

**CREATING A CULTURE OF LIFELONG PREVENTIVE HEALTHCARE:
BETTER CARE FOR CATS**
Margie Scherk DVM, Dip ABVP (feline practice)
catsINK, Vancouver, Canada

To download the electronic version of the in-depth as well as the life-stage (simplified) protocols, please visit <http://www.cathealthy.ca/protocols/>

Dermatology 101: A Pattern Approach to Clinical Dermatology

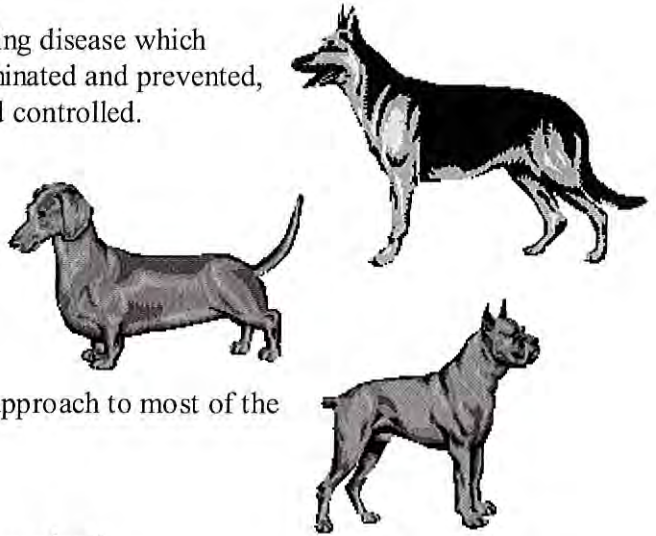
What are the infections? and Why are they there?

Keith A. Hnilica DVM, MS, DACVD University of Tennessee, Knoxville Tn

Almost all dermatology patients have a primary/underlying disease which causes secondary infections. The infections must be eliminated and prevented, but will recur unless the primary disease is identified and controlled.

Most skin cases seen in practice can be successfully managed if these 2 question can be answered. Once the etiology of a patients dermatosis is known, it is a simple matter of therapeutic followthrough to resolve the problem.

The recognition of the basic patterns allows a practical approach to most of the common skin diseases.



10 Clinical Patterns

What are the infections? *(Always secondary)*

1. Folliculitis
2. Pododermatitis
3. Otitis
4. Yeast Dermatitis

Why are they there? *(The key to preventing relapse of infections)*

5. Pruritus
6. Nonpruritic Alopecia (endocrine)
7. Autoimmune Skin Disease
8. Keratinization Defects
9. Lumps, Bumps, and Draining Tracts
10. Weirdopathies

Case example: 2 year old male Labrador that has seasonal pruritus (foot licking) and a moth-eaten hair coat.

What are the Infections?

- Folliculitis

pyoderma, demodex, dermatophyte

- Pododermatitis

bacterial, yeast

- Otitis

bacterial, yeast

- Yeast dermatitis

Why are they there?

Allergies

- Atopy

- Food allergy

- Scabies

Endocrinopathy

- Hypothyroidism

- Cushing's

WHAT IS MAKING MY DOG SO ITCHY?

Evaluation Form

A thorough history can help us find the source of your dog's itching more quickly. Please answer the following questions to help guide the diagnostic process.

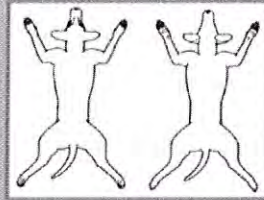
Date _____ Pet owner name _____

Name of dog _____ Age _____ Breed _____ Weight _____

PHYSICAL EVALUATION

Please check any that describe your dog and circle problem areas on the drawing.

- Hair loss
- Foul odor
- Inflammation or redness
- Itching/Scratching
- Otitis (ear infections)
- Licking/Chewing
- Skin lesions (sores)
- Changes in skin (reddish brown stains, discolorations and/or areas that are thick and leathery)
- Other _____



CIRCLE PROBLEM AREAS
(Itching, hair loss, lesions, etc.)

- Has your dog ever had ear problems? Yes No
- Does your dog have any chronic gastrointestinal signs like diarrhea or vomiting? Yes No

SEVERITY EVALUATION

 On a scale of 0 to 10 rank the severity of your dog's symptoms.

SEVERITY OF CONDITION OVERALL

0 1 2 3 4 5 6 7 8 9 10
No symptoms Severe

SEVERITY OF SKIN LESIONS

0 1 2 3 4 5 6 7 8 9 10
No lesions Severe

SEVERITY OF SCRATCHING/LICKING/CHEWING

0 1 2 3 4 5 6 7 8 9 10
No signs Severe

ONSET AND SEASONALITY EVALUATION

- Is this the first time your dog has experienced these symptoms? Yes No
 - If no, at what age did the symptoms first occur? <1 yr 1-3 yrs 4-7 yrs 7+ yrs
 - If no, has it occurred around the same time of year each time? Yes No
 - If no, approximate time of year symptoms occur. _____
- How long have the current symptoms been going on? _____
- Did the itch start gradually and over time become worse? Yes No
- Did the itch come on suddenly without warning? Yes No
- Was there a "rash" first or itching first? Or simultaneous? Rash first Itch first Simultaneous

PARASITE CONTROL

- Is your dog on a flea/heartworm preventative? Yes No
 - If yes, what product(s)? _____
- What months do you administer the preventative? _____
- When was the last time you administered the parasite control? _____

LIFESTYLE EVALUATION

- Where does your dog live? Indoors Outdoors Both
 - If outdoors, please describe environment: _____
- Are there other pets in your household? Yes No
 - If yes, do these pets have the same symptoms? Yes No
 - If these pets are cats, do they go outside? Yes No
- Do you board your dog, take him or her to obedience school, training or groomers? Yes No
 - If yes, when was the last time you took your dog? _____
- Have you taken your dog on a trip to another location? Yes No
 - If yes, please indicate when and location: _____
- Have you recently moved? Yes No
- Have you been to a new dog park or walking trail? Yes No
- Have you used any new shampoo or topical skin treatments recently? Yes No
- Are any humans in your household exhibiting signs? Yes No

DIETARY EVALUATION

- What pet food are you feeding? _____
- Do you feed the same food all the time or provide a variety? Always same Variety
- Have you changed his or her diet recently? Yes No
- Do you give your dog packaged treats? Yes No
- Do you feed your dog "human" food? Yes No

RELATIONSHIP/BEHAVIORAL EVALUATION

Indicate if and how your dog's itching has affected his/her behavior and relationship with you. (CIRCLE ALL APPROPRIATE ANSWERS)

SLEEPS THROUGH THE NIGHT

Always Usually Occasionally Never

ACTIVITY LEVEL

Inactive Much less active Somewhat less active No change

SOCIAL BEHAVIOR

Unsocial A lot less social Somewhat less social No change

RELATIONSHIP CHANGES

Fewer walks No longer sleeps in bed/same room Interacts less with family

PRIOR TREATMENTS

- Has your dog been treated for itching before? Yes No
- Indicate previous treatments administered to your dog: (CHECK ALL THAT APPLY)
 - Steroids Shampoos Sprays Ointments Antibiotics Hypoallergenic food
 - Essential fatty acids Antihistamines Immunotherapy
 - Other (PLEASE SPECIFY) _____

Next Steps

Physical Exam:

A thorough physical evaluation of your dog will help us identify obvious problems and conditions like parasites.

Laboratory Testing:

Ear Swab – To identify any infections in the ear including yeast and/or bacteria.

Skin Scrape/Hair Pluck – To detect scabies or demodex mites.

Impression Smear/Tape Prep – To detect other parasites and check for presence of yeast and/or bacteria.

DERMATOLOGY WORK-UP

SEVERITY OF ITCHING

1 2 3 4 5 6 7 8 9 10
Minor Severe

PET'S NAME: _____

1 WHAT ARE THE INFECTIONS?

Perform 3-Slide Technique™ during the physical exam on multiple sites/lesions.

Slide 1 Skin Scrape (hairplucks): _____ Positive for _____ / _____ Negative

Slide 2 Ear Swab: _____ Positive for _____ / _____ Negative

Slide 3 Tape Prep/Impression Smear: _____ Positive for _____ / _____ Negative

- Pyoderma
- Demodex
- Dermatophytosis
(if suspected, confirm with DTM culture)
- Otitis (Cocci, Yeast, Pseudomonas)
- Pododermatitis (Cocci, Yeast)
- Yeast Dermatitis

2 COMMON ALLERGIC SIGNS¹

A. LUMBAR DERMATITIS

Flea Allergy: (very reliable pattern)

1. Caudal 1/3 of body
2. Flea comb identifying fleas or flea dirt
3. Multiple animals involved or humans affected
4. Variable response to steroids
5. Fall and Spring are often worse but can be year-round

B. EAR-SCRATCH TEST

Scabies: (1-2 are highly reliable)

1. Positive pinnal pedal reflex is 80% diagnostic
2. Ear margin, distal legs, lateral elbow, ventrum
3. Variable responsive to steroids
4. Confirmed by response to treatment
5. Skin Scrapes are often falsely negative

C. PERIANAL DERMATITIS

Food Allergy: (less common but 1-5 increase probability)

1. Perianal dermatitis
2. GI symptoms; more than 3 BM/day, diarrhea, vomiting, flatulence
3. Less than 1 year or older than 5 years at onset
4. Labradors and German Breeds may be predisposed
5. Variable response to steroids

D. FOOT LICKING

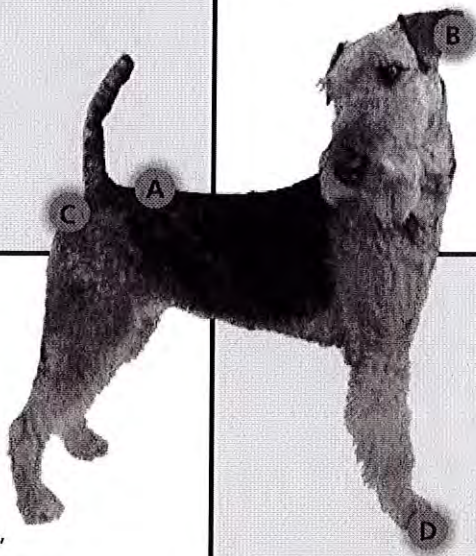
Atopic Dermatitis:

(1-5 are highly reliable)

1. Started at 6 months–3 years of age
2. Front feet affected
3. Inner ear pinnae erythema
4. Lives indoors
5. Ruling out Scabies (ear margin dermatitis) and Flea allergy (lumbar dermatitis)
6. Seasonal symptoms progressing to year-round

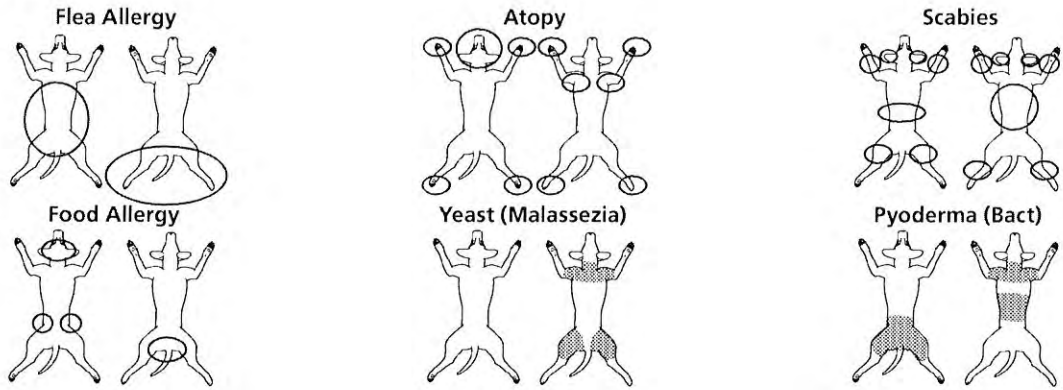
Hypothyroidism: (can mimic allergic dermatitis)

1. Recurrent infection may cause pruritus
2. Lethargy, weight gain, dry coat, hypotrichosis
3. Nonpruritic when infections are resolved



2

PATTERN RECOGNITION



3

TREAT THE ACUTE FLARES:

Cause	Recommended Treatment
Bacterial Pyoderma	
Yeast Infections	
Otitis	
Flea Infestation	
Scabies Treatment	
Steroid "Crisis" Therapy Topical Short-Term Steroid	

TREATMENT, CONTROL AND PREVENTION OF FUTURE FLARES:

Cause	Recommended Treatment
Atopy	Immunotherapy Allergy Vaccine
	Atopica® (Cyclosporine capsules, USP) MODIFIED
Thyroid Supplementation bid	

AVOIDING THE TRIGGERS:

Cause	Treatment	Recommended Treatment
Bacteria Yeast Pollens	Regular bath with an antimicrobial shampoo. Wipe off affected areas (feet, face, etc.) as often as possible	
Otitis	Routine Ear Treatment/cleaning	
Flea and Intestinal Parasites	Year-round Prevention	
Food Triggers	Restricted diet	
House Dust Mites	Dehumidify, replace dog bed, anti-allergy spray (benzyl benzoate)	

PROMOTE SKIN HEALTH AND RESTORE BARRIER FUNCTION:

Cause	Recommended Treatment
Essential Fatty Acids	
Antihistamines	
Soothing, Leave on Conditioner	

RECHECK APPOINTMENT: _____

¹Source: Keith Hnilica, DVM, MS, DACVD.
²Source: R.S. Mueller DipACVD, FACVSc, S.V. Bettenay BVSc, FACVSc, and M. Shipstone BVSc, DipACVD, FACVSc: Value of the pinnal-pedal reflex in the diagnosis of canine scabies, *The Veterinary Record*, Vol 148, Issue 20, 621-623.
³Source: The ACVD task force on canine atopic dermatitis (XIV): clinical manifestations of canine atopic dermatitis, 2001.
⁴Source: Craig Griffin, DVM, DACVD.
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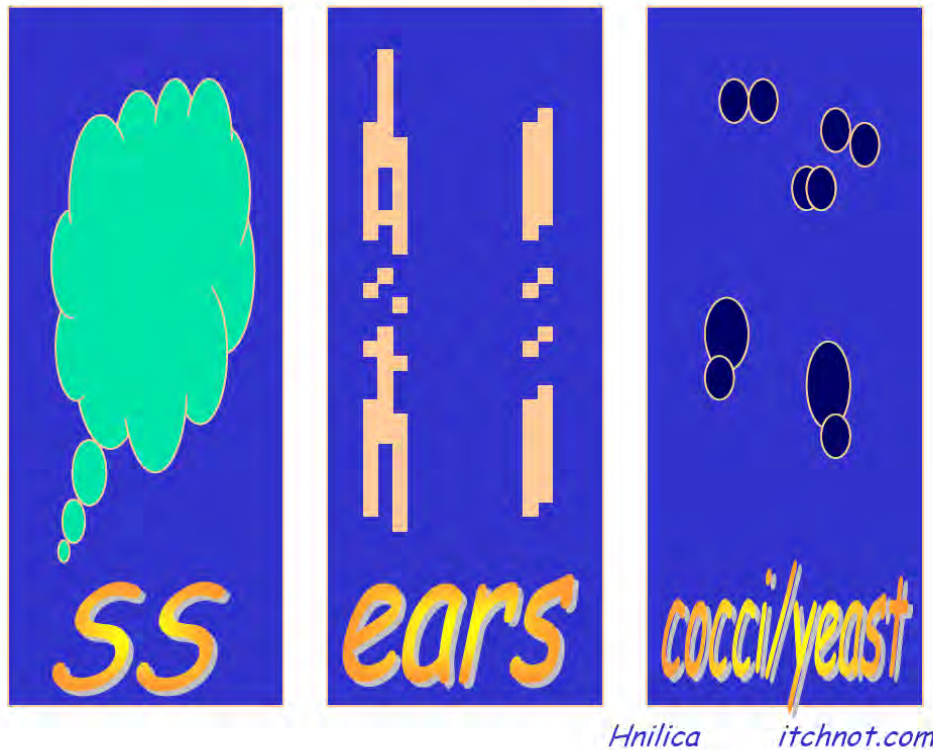
Infectious Disease Diagnosis and Management

Diagnostic Testing

The dermatologic diagnostic minimum database includes skin scrapes, otic swabs, and cutaneous cytology. The goal should be to identify all secondary infections (e.g., pyoderma, demodicosis, dermatophytosis, otitis, *Malassezia* dermatitis, infectious pododermatitis), then formulate a diagnostic plan for identifying and controlling the underlying/primary disease (i.e., allergies, endocrinopathies, keratinization defects, and autoimmune skin diseases).

Ask yourself, “What are the infections?” for every dermatitis cases every time you evaluate the patient.

Using diarrhea and the microscopic fecal exam as a comparison works well since both skin cytology and fecal exams involve the use of a microscope, can easily identify the type of infection, and can be performed by trained technical staff. So why does your clinic perform fecal exams? When is a fecal exam performed (before the doctor’s examination)? Who performed the fecal? Does the clinic charge for the fecal exam? The answers to these questions should be the same for skin cytology (skin scrapings, impression smears, tape preps, and otic swabs).



The practical solution and the best method to answer the question, “What are the infections?” is to implement a minimum data base “infection screening” procedure performed by the technician before the veterinarian examines the patient. Every dermatology patient should have an otic cytology, skin cytology (either an impression smear or tape prep), and a skin scrape performed every time the patient is examined (initially and at every recheck visit). This 3 Slide Technique™ can easily be performed and interpreted by a technician prior to the doctor’s evaluation; exactly how diarrhea cases and fecal exams are handled in most clinics.

Skin Scrapes (Slide #1 in the 3 Slide Technique)

Skin scrapes are the most common dermatologic diagnostic tests. These relatively simple and quick tests can be used to identify many types of parasitic infections. Although they are not always diagnostic, their relative ease and low cost make them essential tests in a dermatologic diagnostic minimum database.

Many practitioners reuse scalpel blades when performing skin scrapes; however, this practice should be stopped because of increased awareness of transmittable diseases (e.g., *Bartonella*, *Rickettsia*, feline leukemia virus [FeLV], feline immunodeficiency virus [FIV], herpes, papillomavirus).

Procedure

Deep Skin Scrapes (for *Demodex* spp except *D. gatoi*). A dulled scalpel blade is held perpendicular to the skin and is used with moderate pressure to scrape in the direction of hair growth. If the area is covered with hair (usually, alopecic areas caused by folliculitis are selected), it may be necessary to clip a small window to access the skin. After several scrapes, the skin should become pink, with the capillaries becoming visible and oozing blood. This ensures that the material collected comes from deep enough within the skin to allow the collection of follicular *Demodex* mites. Most people also squeeze (pinch) the skin to express the mites from deep within the follicles into a more superficial area, so that they may be more easily collected. If the scraping fails to provide a small amount of blood, then the mites may have been left in the follicle, resulting in a false-negative finding. In some situations (with Shar peis or deep inflammation with scarring), it may be impossible to scrape deeply enough to harvest *Demodex* mites. These cases are few in number but require biopsy for identification of the mites within the hair follicles. Hair-plucks from an area of lesional skin may be used to help find mites, but the accuracy of this technique compared with skin scrapes is unknown.

Regardless of the collection technique used, the entire slide should be searched for mites with the use of low power (usually a 10X objective). A search of the entire slide ensures that if only one or two mites are present (as is typical of scabies infection), the user will likely find them. It may be helpful to lower the microscope condenser; this provides greater contrast to the mites, thereby enhancing their visibility. (One must be sure to raise the condenser before looking for cells or bacteria on stained slides.)

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There is no excuse for mistreating a patient who has demodicosis. Lesions caused by demodicosis can look identical to folliculitis lesion caused by bacterial pyoderma and dermatophytosis. Clinical appearance is not an acceptable method to rule-in or rule-out demodicosis. By having the technicians perform a skin scrape as part of the infection screen, 3 Slide Technique™, demodicosis can easily and accurately be identified and treated.

Cutaneous Cytology (Slide #2 in the 3 Slide Technique)

Cutaneous cytology is the second most frequently employed dermatologic diagnostic technique. Its purpose is to help the practitioner to identify bacterial or fungal organisms (yeast) and assess the infiltrating cell types, neoplastic cells, or acantholytic cells (typical of pemphigus complex).

The infections are always secondary to a primary disease; however, all too often, the patient is not evaluated or treated for the primary disease. This is due to 3 predominant factors: treating only the secondary infections over and over, the confusing nature of allergy, and access to cheap steroids which have delayed repercussions.

Superficial Pyoderma (superficial bacterial folliculitis)

Features

Superficial pyoderma is a superficial bacterial infection involving hair follicles and the adjacent epidermis. The infection is almost always secondary to an underlying cause; allergies and endocrine disease are the most common causes (Box 3-3). Superficial pyoderma is one of the most common skin diseases in dogs but rare in cats.

Superficial pyoderma is characterized by focal, multifocal, or generalized areas of papules, pustules, crusts, and scales, epidermal collarettes, or circumscribed areas of erythema and alopecia that may have hyperpigmented centers. Short-coated dogs often present with a “moth-eaten” patchy alopecia, small tufts of hair that stand up, or reddish brown discoloration of white hairs. In long-coated dogs, symptoms can be insidious and may include a dull, lusterless hair coat, scales, and excessive shedding. In both short- and long-coated breeds, primary skin lesions are often obscured by remaining hairs but can be readily appreciated if an affected area is clipped. Pruritus is variable, ranging from none to intense levels. Bacterial infections secondary to endocrine disease may cause pruritus, thereby mimicking allergic skin disease.

Staphylococcus pseudintermedius (previously *Staphylococcus intermedius*) is the most common bacterium isolated from canine pyoderma and is usually limited to dogs. *Staphylococcus schleiferi* is a bacterial species in dogs and humans that is emerging as a common canine isolate in patients with chronic infections and previous antibiotic exposure. Both *Staphylococcus pseudintermedius* and *Staphylococcus schleiferi* may develop methacillin-resistance especially if subtherapeutic doses of antibiotics or fluoroquinolone antibiotics have been previously used in the patient. Additionally, methicillin-resistant *Staphylococcus aureus* (human MRSA) is becoming more common among veterinary species. All three *Staphylococcus* may be zoonotic, moving from humans to canines or from canine to human; immunosuppressed individuals are at most risk.

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Causes of Secondary Superficial and Deep Pyoderma

- Demodicosis, scabies, *Pelodera*
- Hypersensitivity (e.g., atopy, food, flea bite)
- Endocrinopathy (e.g., hypothyroidism, hyperadrenocorticism, sex hormone imbalance, alopecia X)
- Immunosuppressive therapy (e.g., glucocorticoids, progestational compounds, cytotoxic drugs)
- Autoimmune and immune-mediated disorders
- Trauma or bite wound
- Foreign body
- Poor nutrition

Treatment and Prognosis

1. The underlying cause must be identified and controlled.
2. Systemic antibiotics (minimum 3-4 weeks) should be administered and continued 1 week beyond complete clinical and cytological resolution (see Box 3-1).
3. Concurrent bathing every 2 to 7 days with an antibacterial shampoo that contains chlorhexidine or benzoyl peroxide is helpful.
4. If lesions recur within 7 days of antibiotic discontinuation, the duration of therapy was inadequate and antibiotics should be reinstated for a longer time period and better attempts to identify and control the underlying disease should occur.
5. If lesions do not completely resolve during antibiotic therapy or if there is no response to the antibiotics, antibiotic resistance should be assumed and a bacterial culture and sensitivity submitted.
7. If antibiotic resistance is suspected or confirmed, frequent bathing (up to daily) and the frequent application of topical chlorhexidine solutions combined with the simultaneous administration of two different class antibiotics at high doses seem to produce the best results. Monitoring the infection with cytology and cultures with antibiotic sensitivities is important to determine when the treatments can be stopped. Premature discontinuation of therapy, not completely controlling the primary disease, and the use of fluoroquinolone antibiotics will likely perpetuate the resistant infection.
8. The prognosis is good if the underlying cause can be identified and corrected or controlled.

Author's Note:

- ** Superficial Pyoderma is one of the most common skin diseases in dogs and almost always has an underlying cause (allergies or endocrine disease).
- ** Cefpodoxime, Ormetoprim/sulfadimethoxine (Primor), and Convenia provide the most consistent compliance which seem to help reduce the development of resistance when used at high doses.
- ** MRSA, MRSS, MRSI, and MRSP are becoming an emerging problem in some regions of the US.
 - >>The most likely risk factors include previous exposure to fluoroquinolone antibiotics, sub-therapeutic antibiotic dosing, and concurrent steroid therapy.
 - >> Daily baths and topical treatments can be very beneficial in the resolution of the infection.

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>> Maximize the dose of antibiotics and consider using two antibiotics simultaneously to protect additional resistance from developing.

>> Practice good hygiene (HAND WASHING) to prevent zoonosis.

** Consider screening dogs who visit the elderly or sick to prevent zoonosis. Cultures from the nose, lips, ears, axilla, and perianal areas are best for screening patients for MRS.

Malasseziasis (*Malassezia dermatitis*)

Features

Malassezia pachydermatis is a yeast that is normally found in low numbers in the external ear canals, in perioral areas, in perianal regions, and in moist skin folds. Skin disease occurs in dogs when a hypersensitivity reaction to the organisms develops, or when there is cutaneous overgrowth. In dogs, *Malassezia* overgrowth is almost always associated with an underlying cause, such as atopy, food allergy, endocrinopathy, keratinization disorder, metabolic disease, or prolonged therapy with corticosteroids. In cats, skin disease is caused by *Malassezia* overgrowth that may occur secondary to an underlying disease (e.g., feline immunodeficiency virus, diabetes mellitus or an internal malignancy). In particular, generalized *Malassezia* dermatitis may occur in cats with thymoma-associated dermatosis or paraneoplastic alopecia. Malasseziasis is common in dogs, especially among West Highland White terriers, Dachshunds, English setters, Basset hounds, American cocker spaniels, Shih tzus, Springer spaniels, and German shepherds. These breeds may be predisposed. Malasseziasis is rare in cats.

Moderate to severe pruritus is seen, with regional or generalized alopecia, excoriations, erythema, and seborrhea. With chronicity, affected skin may become lichenified, hyperpigmented, and hyperkeratotic (leathery or elephant-like skin). An unpleasant body odor is usually present. Lesions may involve the interdigital spaces, ventral neck, axillae, perineal region, or leg folds. Paronychia with dark brown nail bed discharge may be present. Concurrent yeast otitis externa is common.

Diagnosis

1. Rule out other differentials
2. Cytology (tape preparation, impression smear): yeast overgrowth is confirmed by the finding round-to-oval, budding yeasts per high power field (100 \times). In yeast hypersensitivity, organisms may be difficult to find

Treatment and Prognosis

1. Any underlying cause (allergies, endocrinopathy, keratinization defect) must be identified and corrected.
2. For mild cases, topical therapy alone is often effective. The patient should be bathed every 2 to 3 days with shampoo that contains 2% ketoconazole, 1% ketoconazole/2% chlorhexidine, 2% miconazole, 2% to 4% chlorhexidine, or 1% selenium sulfide (dogs only). Shampoos that have

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two active ingredients provide better efficacy. Treatment should be continued until the lesions resolve and follow-up skin cytology reveals no organisms (approximately 4 weeks).

3. The treatment of choice for moderate to severe cases is ketoconazole (dogs) or fluconazole 10mg/kg PO with food every 24 hours, Treatment should be continued until lesions resolve and follow-up skin cytology reveals no organisms (approximately 4 weeks).
4. Alternatively, treatment with terbinafine 5-40mg/kg PO every 24 hours or itraconazole (Sporonox) 5-10mg/kg every 24 hours for 4 weeks may be effective.
5. Pulse therapy protocols have been published using several drugs and a variety of schedules; however, these often take longer to resolve the active infection.
5. The prognosis is good if the underlying cause can be identified and corrected. Otherwise, regular once- or twice-weekly antiyeast shampoo baths may be needed to prevent relapse. This disease is not considered contagious to other animals or to humans, except for immunocompromised individuals.

Authors' Note:

- ** Yeast dermatitis is currently the most commonly missed diagnosis in US general practices. Any patient with leathery, elephant-skin like lesions on the ventrum should be suspected of having *Malassezia dermatitis*.
- ** Cutaneous cytology is not always successful for finding *Malassezia* organisms requiring the clinician to rely on clinical lesion patterns to make a tentative diagnosis.
- ** Yeast dermatitis is severely pruritic with owners reporting an itch level of 10 on a 0-10 visual analog scale.

Canine Generalized Demodicosis

Features

Canine generalized demodicosis may appear as a generalized skin disease that may have genetic tendencies and can be caused by three different species of demodectic mites: *D. canis*, *D. injai*, and an unnamed short-bodied *Demodex* mite. *D. canis*, a normal resident of the canine pilosebaceous unit (hair follicle, sebaceous duct, and sebaceous gland), is primarily transmitted from the mother to neonates during the first 2 to 3 days of nursing, but adult-to-adult transmission may rarely occur. *D. injai*, a recently described, large, long-bodied *Demodex* mite, is also found in the pilosebaceous unit, but its mode of transmission is unknown. Mode of transmission is also unknown for the short-bodied unnamed *Demodex* mite, which, unlike the other two species, lives in the stratum corneum. Depending on the dog's age at onset, generalized demodicosis is classified as juvenile-onset or adult-onset. Both forms are common in dogs. Juvenile-onset generalized demodicosis may be caused by *D. canis* and the short-bodied unnamed *Demodex* mite. It occurs in young dogs, usually between 3 and 18 months of age, with highest incidence in medium-sized and large purebred dogs. Adult-onset generalized demodicosis can be caused by all three mite species and occurs in dogs older than 18 months of age, with highest incidence in middle-aged to older dogs that are immunocompromised because of an underlying condition such as endogenous or iatrogenic hyperadrenocorticism, hypothyroidism, immunosuppressive drug therapy, diabetes mellitus, or neoplasia. To date, only adult-onset disease has been reported with *D. injai*, with highest incidence noted in terriers.

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Clinical signs of infestation with either *D. canis* or the unnamed *Demodex* mite are variable. Generalized demodicosis is defined as five or more focal lesions, or two or more body regions affected. Usually, patchy, regional, multifocal, or diffuse alopecia is observed with variable erythema, silvery grayish scaling, papules, or pruritus. Affected skin may become lichenified, hyperpigmented, pustular, eroded, crusted, or ulcerated from secondary superficial or deep pyoderma. Lesions can be anywhere on the body, including the feet. Pododemodicosis is characterized by any combination of interdigital pruritus, pain, erythema, alopecia, hyperpigmentation, lichenification, scaling, swelling, crusts, pustules, bullae, and draining tracts. Peripheral lymphadenomegaly is common. Systemic signs (e.g., fever, depression, anorexia) may be seen if secondary bacterial sepsis develops.

D. injai infestations are typically characterized by a focal areas of greasy seborrhea (seborrhea oleosa), especially over the dorsum of the trunk. Other skin lesions may include alopecia, erythema, hyperpigmentation, and comedones. Small breeds and terriers seem to predisposed to *Demodex injai* infections.

Diagnosis

1. Microscopy (deep skin scrapes): many demodectic adults, nymphs, larvae, and ova are typically found with *D. canis* and the short-bodied, unnamed demodectic mite, although *D. canis* may be difficult to find in fibrotic lesions and in feet. With *D. injai*, mites may be low in number and difficult to find requiring skin biopsies.

Treatment and Prognosis

1. If adult-onset, any underlying conditions should be identified and corrected. All steroid containing therapies should be discontinued as steroid administration is the mostcommon cause of adult onset demodicosis.
2. Intact dogs, especially females, should be neutered. Estrus or pregnancy may trigger relapse.
3. Any secondary pyoderma should be treated with appropriate long-term (minimum 3-4 weeks) systemic antibiotics that are continued at least 1 week beyond clinical resolution of the pyoderma.
4. Topical shampoo therapy using a 1-3% benzoyl peroxide shampoo every 3-7 days will help speed resolution and enhance the mitacidal treatments.
5. Effective Mitacidal therapies include the following:
 - *Ivermectin 0.2-0.6mg/kg PO every 24 hours is often effective against generalized demodicosis. Initially, ivermectin 0.1mg/kg PO is administered on day 1, then 0.2mg/kg PO is administered on day 2, with oral daily increments of 0.1mg/kg until 0.2-0.6mg/kg/day is being administered, assuming that no signs of toxicity develop. The cure rate for 0.4mg/kg/day ivermectin is 85% to 90%.
 - *Milbemycin oxime, 0.5 to 2mg/kg PO every 24 hours. The cure rate is 85% to 90%.
 - *Doramectin is also reported to be effective against canine demodicosis at a dose of 0.6mg/kg SC once weekly. The cure rate is approximately 85%. Adverse effects are uncommon but include, as for ivermectin, dilated pupils, lethargy, blindness, and coma.
 - *For dogs ≥ 20 kg, the use of 9% amitraz collars may be effective. In small dogs, use of 9% amitraz collars alone may be as effective as ivermectin (0.6mg/kg/day PO).

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* Topical application of Promeris (topical metaflumizone and amitraz solution) (topical metaflumizone and amitraz solution) every two weeks has demonstrated good efficacy.

*Moxidectin has demonstrated variable efficacy when applied every 2-4 weeks.

Historical Treatment Include:

Traditional miticidal treatment entails the following:

- Total body hair coat clip if dog is medium- to long-haired
 - Weekly bath with 2.5% to 3% benzoyl peroxide shampoo, followed by a total body application of 0.03% to 0.05% amitraz solution. The cure rate ranges from 50% to 86%.
 - * For demodectic pododermatitis, in addition to weekly amitraz dips, foot soaks in 0.125% amitraz solution should be performed every 1 to 3 days.
6. Regardless of the miticidal treatment chosen, therapy is administered over the long term (weeks to months). Treatments should be continued for at least 1 month beyond the time when the first follow-up skin scrapings becomes negative for mites (total of two negative skin scrapings).
 7. The prognosis is good to fair. Relapses may occur, requiring periodic or lifelong treatment in some dogs. The use of glucocorticosteroids in any dog that has been diagnosed with demodicosis should be avoided. Because of its hereditary predisposition, neither female nor male dogs with juvenile-onset generalized demodicosis should be bred. *D canis* is not considered contagious to cats or to humans. It is transmitted from bitch to newborn puppies during the first 2 to 3 days of nursing, and possibly between adult dogs that are close cohabitants. The mode of transmission for *D injai* and the unnamed short-bodied *Demodex* mite is unknown.

Author's Note:

Steroids are the most common cause of adult onset Demodicosis.

Products containing amitraz tend to be the most toxic usually due to the product vehicle.

Aggressive treatment should be tried for up to six months before giving up.

One of the most common causes of treatment failure is that the patient will look greatly improved before negative skin scrapes are achieved. Many owners will discontinue treatment prematurely resulting in relapse.

The average time to achieve clinical improvement is 4-6 weeks; the first negative skin scrape usually occurs around 6-8 weeks; and most patients need approximately 3 months of treatment to resolve the infection based on two negative skin scrapes at least 3 weeks apart.

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Diagnosing and Treating Canine Allergy

Canine Atopy (environmental, pollen allergies)

Features

Canine atopy is a hypersensitivity reaction to inhaled (possibly a historic theory) or cutaneously absorbed environmental antigens (allergens) in genetically predisposed individuals. It is common in dogs, with age of onset ranging from 6 months to 6 years. However, in most atopic dogs, symptoms first appear at between 1 and 3 years of age.

Symptoms begin as skin erythema and pruritus (licking, chewing, scratching, rubbing), which may be seasonal or nonseasonal, depending on the offending allergen. The distribution of the pruritus usually involves the **feet**, flanks, groin, axillae, face, and ears. Self-trauma often results in secondary skin lesions, including salivary staining, alopecia, excoriations, scales, crusts, hyperpigmentation, and lichenification. Secondary pyoderma, *Malassezia* dermatitis, and otitis externa are common. Chronic acral lick dermatitis, recurrent pyotraumatic dermatitis, conjunctivitis, hyperhidrosis (sweating), and, rarely, allergic bronchitis or rhinitis may be seen.

Top Differentials

Differentials include food allergy, scabies, *Malassezia* dermatitis, bacterial pyoderma, as well as other hypersensitivities (flea bite, contact), parasites (cheyletiellosis, pediculosis), and folliculitis (dermatophyte, *Demodex*).

Diagnosis

1. Seasonal foot-licking is the most unique and typical symptom of atopy. If year-round allergens (house dust mites) are causing the allergy, the foot-licking may be nonseasonal.
2. Allergy testing (intradermal, serologic): allergy testing can be highly variable according to the method used. Positive reactions to grass, weed, tree, mold, insect, dander, or indoor environmental allergens are seen. False-negative and false-positive reactions may occur.
3. Dermatohistopathology (nondiagnostic): superficial perivascular dermatitis that may be spongiotic or hyperplastic. Inflammatory cells are predominantly lymphocytes and histocytes. Eosinophils are uncommon. Neutrophils or plasma cells suggest secondary infection.

Treatment and Prognosis

1. **Infection Prevention:**
 - a. Any secondary pyoderma, otitis externa, and *Malassezia* dermatitis should be treated with appropriate therapies. Controlling and preventing secondary infection is an essential component of managing atopic dogs. Bathing every 3 – 7 days and treating the ears after every bath helps wash off pollens and disinfect the skin and ear canals, preventing the secondary infections from recurring.

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2. Symptomatic Therapy (itch control):

a An integrated flea control program should be instituted to prevent flea bites from aggravating the pruritus.

b Topical therapy with antimicrobial shampoos and anti-itch conditioners, and sprays (i.e., those containing oatmeal, pramoxine, antihistamines, or glucocorticoids) applied every 2 to 7 days or as needed may help reduce clinical symptoms.

C Systemic antihistamine therapy reduces clinical symptoms in many cases (Table 7-1). Antihistamines can be used alone or in combination with glucocorticoids or essential fatty acids for a synergistic effect. One- to two-week long therapeutic trials with different antihistamines may be required to determine which is most effective.

D Oral essential fatty acid supplements (180mg EPA/10 lb) help control pruritus in 20% to 50% of cases, but 8 to 12 weeks of therapy may be needed before beneficial effects are seen. Also, a synergistic effect is often noted when essential fatty acid supplements are administered in combination with glucocorticoids or antihistamines.

E Dextromethorphan, an opioid antagonist, may also be a useful adjunct in managing the licking, chewing, and biting behaviors associated with allergic dermatitis in dogs. Dextromethorphan 2mg/kg PO should be administered every 12 hours. A beneficial effect should be seen within 2 weeks.

F Systemic glucocorticoid therapy is often effective (75%) in controlling pruritus but almost always result in adverse effects ranging to mild (PU/PD) to severe (immuno-dysfunction, Demodicosis, and calcinosis cutis). It is a therapeutic option if the allergy season is very short but may result in unacceptable adverse effects, especially if used over the long term.

1. Potent, long acting injectable steroids are contraindicated for the treatment of allergies due to their comparatively short anti-inflammatory benefits (3 weeks) relative to the prolonged metabolic and immuno-depressive effects (6-10 weeks).

2. Injectable short acting steroids (dexamethasone Sodium Phosphate, 0.5 – 1 mg/kg or prednisilone acetate 0.1mg – 1 mg/kg) are effective at providing relief and may last 2 to 3 weeks if there are no concurrent secondary infection. This treatment option allows the clinician to more closely control and monitor the patients steroids use compared to oral treatments that are administered by the owner.

3. Temaril-P (trimeprazine and prednisilone combination) is a unique drug that provides significant antipruritic effects at a relatively lower dose of the prednisilone. One tablet should be administered per 10 to 20 kg every 24 to 48 hours. The dosage should be tapered to the lowest possible dose and frequency.

4. Prednisone 0.25 to 1mg/kg (or methylprednisolone 0.2-0.8mg/kg) PO should be administered every 24 to 48 hours for 3 to 7 days. The dosage should be tapered to the lowest possible dose and frequency.

5. All dogs treated with long-term steroids (more than 3 months) should be frequently monitored for liver disease and UTI.

8. Allergy Treatment (immune-modulation)

a. Exposure to offending allergens should be reduced, if possible, by their removal from the environment. High-efficiency particulate (HEPA) air and charcoal filters should be used to reduce pollens, molds, and dust in the home. For house dust mite-sensitive dogs, household treatments for carpets, mattresses, and upholstery with the acaricide benzyl benzoate once a month for approximately 3 months, then every 3 months thereafter, may effectively eliminate house dust mites from the environment. Old dog beds should be discarded as these accumulate house dust mite antigens. Dehumidifying the house to below 40% relative

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humidity decreases house dust mite, mold, and flea antigen loads. To achieve this, high-efficiency dehumidifiers that are capable of pulling several liters of water from the air per day are required.

b. Cyclosporine (Atopica) helps control pruritus in 75% of atopic dogs. A dose of 5mg/kg PO should be administered every 24 hours until beneficial effects are seen (approximately 4-6 weeks). Then, dosage frequency should be tapered down to every 48 to 72 hours. For long-term control, approximately 25% of dogs require daily dosing, 50% can be controlled with every-other-day dosing, and approximately 25% can be controlled with twice-weekly dosing. Glucocorticoids can be used initially to speed response. As of this writing, there are no statistically significant increases in tumor risk or severe infection resulting from the immune effects of cyclosporine.

c. With immunotherapy (allergy vaccine), 60% to 75% of atopic dogs show good (some medical therapy still needed) to excellent (no other therapy needed) response. Clinical improvement is usually noted within 3 to 5 months of initiation of immunotherapy, but it can take up to 1 year in some dogs.

11. The prognosis is good, although lifelong therapy for control is needed in most dogs. Relapses (pruritic flare-ups with/without secondary infections) are common, so individualized treatment adjustments to meet patient needs may be required periodically. In dogs that become poorly controlled, one should rule out secondary infection (e.g., that caused by bacteria or *Malassezia*); sarcoptic mange; demodicosis; concurrent food, flea bite, and recently acquired hypersensitivity to additional environmental allergens. Because a strong genetic component is present, the breeding of any male or female dog with clinical signs of atopic dermatitis should be discouraged.

Box

Author's Note

Our profession has excelled at reducing the use of steroids for arthritis; however, we have failed to make similar achievements for allergic disease including atopy. Since both disease have many similarities, including chronicity and multimodal therapeutic options, our goal should be to minimize the use of steroids for allergic diseases through the use of alternative, safer treatment options. To achieve best medicine, the frequency of steroid use should be similar for patients with arthritis and allergy.

The use of long-acting, injectable steroids should be stopped due to the profound impact on the metabolic and immune systems as well as the growing concern of legal liability for the practitioner.

Author's Note

The only real, long-term options for treating the allergic immune response to environmental allergens are avoidance, allergy vaccine, or cyclosporine (Atopica). Based on typical cgeneral practice demographics, every full time small animal veterinarian should have approximately 20-30 patients who are no longer controlled with symptomatic therapy and need more aggressive treatment (allergy vaccine or cyclosporine).

Canine Food Hypersensitivity

Features

Canine food hypersensitivity is an adverse reaction to a food or food additive. It can occur at any age, from recently weaned puppies to elderly dogs that have been eating the same dog food for years. Approximately 30% of dogs diagnosed with food allergy are younger than 1 year of age. It is common in dogs.

Canine food hypersensitivity is characterized by nonseasonal pruritus that may or may not respond to steroid therapy. The pruritus may be regional or generalized and usually involves the ears, feet, inguinal or axillary areas, face, neck, and **perineum**. Affected skin is often erythematous, and a papular rash may be present. Self-trauma-induced lesions include alopecia, excoriations, scales, crusts, hyperpigmentation, and lichenification. Secondary superficial pyoderma, *Malassezia* dermatitis, and otitis externa are common. Other symptoms that may be seen are acral lick dermatitis, chronic seborrhea, and recurring pyotraumatic dermatitis. Some dogs are minimally pruritic, with the only symptom being recurrent infection with pyoderma, *Malassezia* dermatitis, or otitis. In these cases, the pruritus is present only when secondary infections are left untreated. Occasionally, urticaria or angioedema may occur. Concurrent gastrointestinal signs (e.g., frequent bowel movements, vomiting, diarrhea, flatulence) are reported in 20%-30% of cases.

Top Differentials

Differentials include atopy, scabies, *Malassezia* dermatitis, bacterial pyoderma, as well as other hypersensitivities (flea bite, contact), parasites (cheyletiellosis, pediculosis), and folliculitis (dermatophyte, *Demodex*).

Diagnosis

1. Perianal dermatitis with or without recurrent otitis is the most common and unique feature of food allergy. However, food allergy can manifest in many patterns and should be suspected for atypical pruritic patient including cases of recurrent infections without pruritus.
2. Dermatohistopathology (nondiagnostic): varying degrees of superficial perivascular dermatitis. Mononuclear cells or neutrophils may predominate. Eosinophils may be more numerous than in atopy
3. Food allergy testing (intradermal, serologic)(nondiagnostic): not recommended because test results are unreliable. Some dogs will have positive reactions to storage mite antigens, which may be clinically relevant, or they may be caused by cross-reactivity with other insects. Storage mites are ubiquitous, and their clinical significance is currently unknown.
4. Response to hypoallergenic diet trial: symptoms improve within 10 to 12 weeks of initiation of a strict home-cooked or commercially prepared restricted diet (one protein and one carbohydrate source). The hypoallergenic diet should not contain food ingredients previously administered in dog food, treats, or table scraps, nor should flavored heartworm preventative, flavored medications, nutritional supplements, or chewable treats (i.e., pig ears, cow hooves, rawhide, dog biscuits, table food such as cheese or peanut butter to hide pills in) be

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administered during the hypoallergenic diet trial. Beef and dairy are the most common food allergens in dogs and avoiding these alone may result in clinical improvement. Other common food allergies include chicken, eggs, soy, corn, and wheat.

5. Provocative challenge: recurrence of symptoms within hours to days of reintroduction of suspect allergen into the diet.

Treatment and Prognosis

1. Infection Prevention:

- a. Any secondary pyoderma, otitis externa, and *Malassezia* dermatitis should be treated with appropriate therapies. Controlling and preventing secondary infection is an essential component of managing atopic dogs. Bathing every 3 – 7 days and treating the ears after every bath helps wash off pollens and disinfect the skin and ear canals, preventing the secondary infections from recurring.

2. Symptomatic Therapy (itch control) is variably effective for food allergy:

a An integrated flea control program should be instituted to prevent flea bites from aggravating the pruritus.

b Topical therapy with antimicrobial shampoos and anti-itch conditioners, and sprays (i.e., those containing oatmeal, pramoxine, antihistamines, or glucocorticoids) applied every 2 to 7 days or as needed may help reduce clinical symptoms.

c Systemic antihistamine therapy reduces clinical symptoms in many cases (Table 7-1). One- to two-week long therapeutic trials with different antihistamines may be required to determine which is most effective.

d Oral essential fatty acid supplements (180mg EPA/10 lb) help control pruritus in 20% to 50% of cases, but 8 to 12 weeks of therapy may be needed before beneficial effects are seen. Also, a synergistic effect is often noted when essential fatty acid supplements are administered in combination with glucocorticoids or antihistamines.

e Dextromethorphan, an opioid antagonist, may also be a useful adjunct in managing the licking, chewing, and biting behaviors associated with allergic dermatitis in dogs. Dextromethorphan 2mg/kg PO should be administered every 12 hours. A beneficial effect should be seen within 2 weeks.

f Systemic glucocorticoid therapy is only variably effective (unpredictable minimal to good response) in controlling pruritus cause by the food allergy; but almost always result in adverse effects ranging to mild (PU/PD) to severe (immuno-dysfunction, Demodicosis, and calcinosis cutis). (see Atopy section)

1. Potent, long acting injectable steroids are contraindicated for the treatment of allergies due to their comparatively short anti-inflammatory benefits (3 weeks) relative to the prolonged metabolic and immuno-depressive effects (6-10 weeks).

2. Injectable short acting steroids (dexamethasone Sodium Phosphate, 0.5 – 1 mg/kg or prednisilone acetate 0.1mg – 1 mg/kg) are effective at providing relief and may last 2 to 3 weeks if there are no concurrent secondary infection. This treatment option allows the clinician to more closely control and monitor the patient's steroids use compared to oral treatments that are administered by the owner.

3. All dogs treated with long-term steroids (more than 3 months) should be frequently monitored for liver disease and UTI.

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8. Food Allergy Treatment

- a. Offending dietary allergen(s) should be avoided. A balanced home-cooked diet or a commercial hypoallergenic diet should be provided.
 - b. To identify offending substances to be avoided (challenge phase after food allergy has been confirmed with the dietary trial) one new food item should be added to the hypoallergenic diet every 2 to 4 weeks. If the item is allergenic, clinical symptoms will recur within 7 to 10 days. *Note:* Some dogs (approximately 20%) should be fed home-cooked diets to remain symptom-free. For these dogs, commercial hypoallergenic diets are ineffective, presumably because their hypersensitivity relates to a food preservative or dye.
 - c. Anecdotal reports suggest that higher doses (10mg/kg) of cyclosporine (Atopica) may be beneficial in reducing the allergic immune response and symptoms of food allergy.
8. The prognosis is good. In dogs that are poorly controlled, owner noncompliance should be ruled out, along with development of hypersensitivity to an ingredient in the hypoallergenic diet, secondary infection (caused by bacteria, *Malassezia*, dermatophyte), scabies, demodicosis, atopy, flea allergy dermatitis, and contact hypersensitivity.

Author's Note

Due to recent food industry changes, there has been an explosion of products available through prescription or over-the-counter and the listing is beyond the scope of this text.

Many of the over-the-counter diets are sufficiently restricted and of high enough quality to produce clinical benefit when a food allergic patient restricted to one of the nonBeef and nondairy products.

Food allergy is responsible for most of the very unusual clinical symptom patterns in dogs with recurrent infections (with or without pruritus).

Poor owner compliance should be expected making the long-term management of food allergic patients difficult and frustrating; repeated lapses in diet result in flare-ups in the pruritus and secondary infections.

Author's Note

The use of long-acting, injectable steroids should be stopped due to the profound impact on the metabolic and immune systems as well as the growing concern of legal liability for the practitioner.

Flea Allergy Dermatitis (flea bite hypersensitivity)

Features

Flea allergy dermatitis is a common skin disease in dogs and cats sensitized to flea saliva proteins through repeated and intermittent flea bites. Symptoms are usually seasonal (warm weather months and in the fall) in temperate zones and often nonseasonal in subtropical and tropical areas. Fall is often the most severe season relating to when the first cold snap occurs.

Dogs

The distribution typically involves the caudodorsal lumbosacral area, dorsal tail head, caudomedial thighs, abdomen, and flanks. Lesions include pruritic, papular, crusting eruptions

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with secondary erythema, seborrhea, alopecia, excoriations, pyoderma, hyperpigmentation, and lichenification.

Cats

Cats do not have a pattern unique to flea allergy dermatitis. Patients commonly present with pruritic miliary dermatitis with secondary excoriations, crusting, and alopecia of the neck, dorsal lumbosacral area, caudomedial thighs, and ventral abdomen. Other symptoms include symmetrical alopecia secondary to excessive grooming and eosinophilic granuloma complex lesions.

Top Differentials

Differentials include atopy, food hypersensitivity, other ectoparasites (scabies, cheyletiellosis, pediculosis, demodicosis), superficial pyoderma, dermatophytosis, demodicosis, and *Malassezia* dermatitis.

Diagnosis

1. Lumbar dermatitis in the dog is the most consistent and unique feature of flea allergy dermatitis. In cats, flea allergy should be highly suspected in any cat with skin disease.
2. Visualization of fleas or flea excreta on body: may be difficult on flea-allergic animals as flea-allergic animals are very effective at removing fleas through grooming
3. Allergy testing (intradermal, serologic): positive skin test reaction to flea antigen or positive serum immunoglobulin (Ig)E antiflea antibody titer is highly suggestive, but false-negative results are possible
4. Dermatohistopathology (nondiagnostic): varying degrees of superficial or deep perivascular to interstitial dermatitis, with eosinophils often predominating
5. Response to aggressive flea control (nitenpyram administered every other day for 1 month): symptoms resolve

Treatment and Prognosis

1. Integrated flea management program (insect growth regulator combined with an adulticide combined with environmental treatments) is essential due to the progressive tolerance of the flea to available adulticides. With time, specific active ingredients typically lose efficacy due to the chronic exposure and genetic drift of the flea.
2. Topical or systemic insect growth regulators (lufenuron, piriproxyfen, methoprene) may be effective alone or used in combination with adulticidal therapy.
3. Affected and all in-contact dogs and cats should be treated with adulticidal flea sprays, spot-on solutions, orals, or dips every 7 to 30 days, as instructed on the label. Products that contain spinosid, imidacloprid, selamectin, or fipronil are especially effective when used topically every 2 to 4 weeks. In heavily flea-infested environments, fleas may continue to be found on animals for several months in spite of topical flea control. In these cases, affected animals should also be administered nitenpyram at a minimum dose of 1mg/kg PO every 24 to 48 hours for 2 to 4 weeks, or until fleas are no longer seen. The environment should be treated (see number 5 below).

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4. Flea-allergic animals should be treated prophylactically with nitenpyram, minimum dose 1mg/kg PO, on any day that an encounter is planned with other potentially flea-infested animals (e.g., a visit to the groomer, veterinary hospital, park, another household with animals). No more than one treatment with nitenpyram should be administered per day.
5. In heavily flea-infested environments, areas where pets spend the most time should be treated. Indoor premises should be treated with an insecticide and an insect growth regulator (e.g., methoprene, piriproxyfen). The outdoor environment should be treated with insecticidal or biologic products designed for such use.
6. Flea control therapy should be continued from spring until first snowfall in temperate areas and year-round in warm climates. Year-round flea infestations can be perpetuated indoors and on wildlife despite extreme cold outdoors.
7. **Symptomatic Therapy (itch control):**
 - a Topical therapy with antimicrobial shampoos and anti-itch conditioners, and sprays (i.e., those containing oatmeal, pramoxine, antihistamines, or glucocorticoids) applied every 2 to 7 days or as needed may help reduce clinical symptoms.
 - b Systemic antihistamine therapy reduces clinical symptoms in many cases (Table 7-1).
 - c Systemic glucocorticoid therapy is often effective (75%) in controlling pruritus but almost always result in adverse effects ranging to mild (PU/PD) to severe (immuno-dysfunction, Demodicosis, and calcinosis cutis). It is a therapeutic option if the allergy season is very short but may result in unacceptable adverse effects, especially if used over the long term.
 1. Potent, long acting injectable steroids are contraindicated for the treatment of allergies due to their comparatively short anti-inflammatory benefits (3 weeks) relative to the prolonged metabolic and immuno-depressive effects (6-10 weeks).
 2. Injectable short acting steroids (dexamethasone Sodium Phosphate, 0.5 – 1 mg/kg or prednisilone acetate 0.1mg – 1 mg/kg) are effective at providing relief and may last 2 to 3 weeks if there are no concurrent secondary infection. This treatment option allows the clinician to more closely control and monitor the patients steroids use compared to oral treatments that are administered by the owner.
 3. Temaril-P (trimeprizine and prednisilone combination) is a unique drug that provides significant antipruritic effects at a relatively lower dose of the prednisilone. One tablet should be administered per 10 to 20 kg every 24 to 48 hours. The dosage should be tapered to the lowest possible dose and frequency.
 4. Prednisone 0.25 to 1mg/kg (or methylprednisolone 0.2-0.8mg/kg) PO should be administered every 24 to 48 hours for 3 to 7 days. The dosage should be tapered to the lowest possible dose and frequency.
 5. All dogs treated with long-term steroids (more than 3 months) should be frequently monitored for liver disease and UTI.
8. The prognosis is good if strict flea control is practiced. Fleas may infest other in-contact animals and humans. They may carry blood-borne diseases in a manner similar to ticks.

Author's note

The use of long-acting, injectable steroids should be stopped due to the profound impact on the metabolic and immune systems as well as the growing concern of legal liability for the practitioner.

Any dog with lumbar dermatitis or any cat with skin disease should be highly suspected of having flea allergy dermatitis even if the patient has been treated with seemingly good flea control therapies.

A nitenpyram trial (every other day for 1 month) is the most efficient and cost effective way to convince the owner and yourself of the role of flea allergy in a pruritic patient.

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Feline Dermatology and Pruritus

Feline pruritus is one of the most common dermatologic problems affecting cats. Mild cases often respond to empiric antipruritic treatments. Severe or chronic cases need a thorough workup to identify and control the primary etiology for long-term success. Since many etiologies can cause the 3 common clinical patterns (alopecia, miliary dermatitis, and eosinophilic granuloma complex) a prioritized differential list should be used to systematically work through the different etiologies. Flea allergy, insect hypersensitivity, Demodicosis, and food allergy are the most common pruritic diseases; however, dermatophytosis and other infectious causes of alopecia and miliary dermatitis should be eliminated.

Clinical Features:

Regardless of the underlying cause, cats seem to react with 3 distinct clinical patterns. Alopecia is one of the most common presentations, especially on the abdomen and inner thighs. Often there are no skin lesions just alopecia. Miliary dermatitis and eosinophilic granuloma complex (eosinophilic plaque, linear granuloma, indolent ulcer, oral granuloma) are also common feline dermatologic patterns. Regardless of the clinical pattern, the differential list is similar.

THE NEW ERA: 5 Steps to success WITH OUT steroids

1. **Rule out Flea allergy** is the most common allergy in cats and most pruritic cats respond to an aggressive flea control trial. Cats commonly present with pruritic miliary dermatitis with secondary excoriations, crusting, and alopecia of the neck, dorsal lumbosacral area, caudomedial thighs and/or ventral abdomen. Other symptoms include symmetrical alopecia secondary to excessive grooming, and eosinophilic granuloma complex lesions. Many cats are extremely effective at removing fleas and flea dirt by grooming making it difficult to prove the existence of a flea infestation. Therefore, all pruritic cats should be treated aggressively for possible flea allergy dermatitis. Capstar administered every other day for 1 month effectively prevents flea feeding and eliminates the pet's exposure to flea saliva. If the patient is better after 12-15 every other day doses of Capstar, FLEA exposure and FLEA ALLERGY has been confirmed.
2. **Culture for RING WORM: Dermatophyte** is the most common infectious skin disease in cats. The most common clinical lesion pattern is miliary dermatitis. Diagnosis is usually based on Wood's lamp examination and cultures. Since ringworm can mimic so many other diseases, it should be considered and ruled out in all cats with skin lesions.
3. **Eliminate Mites: Demodex gatoi** may be the more common Demodex species (especially in the Southern states) and causes pruritic symptoms similar to allergic dermatitis. *D. gatoi* is the short bodied mite that inhabits the superficial skin structures and may be contagious to other cats. Generalized disease is characterized by variably pruritic, multifocal, patchy, regional, or symmetric alopecia with or without erythema, scaling, crusts, macules, and hyperpigmentation. Lesions usually involve the head, neck, limbs, flanks, and/or the ventrum. Ceruminous otitis externa and secondary pyoderma may be present. *Notoedres cati* is rare. *Cheyletiella* mites live on hair and fur and are usually found with tape preps, skin scrapes, flea comb surf examination, or fecal.
4. **Consider a food trial if the Patient will comply:** Food allergy is a nonseasonal pruritic dermatitis that may respond to steroids. The distribution of the pruritus and lesions may be localized to the head and neck, or it may be generalized and involve the trunk, ventrum, and limbs. Skin lesions are variable and may include alopecia, erythema, miliary dermatitis,

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eosinophilic granuloma complex lesions, excoriations, crusts, and scales. Ceruminous otitis externa is often present. Concurrent gastrointestinal symptoms may be present.

5. **Cyclosporine Therapy** controls almost all other allergies or other caused of immune mediated dermatosis in cats. Insect hypersensitivity (Mosquitoes, moth, cockroach, ants, etc) is possibly the second most common cause of allergy in cats. Atopy symptoms may be seasonal or non-seasonal depending on the offending allergens. The pruritus may occur on the head, neck, and ears, or it may be in other areas such as the ventral abdomen, caudal thighs, forelegs, and/or the lateral thorax. Pemphigus is usually a nonpruritic disease with lesions that include superficial erosions, crusts, scales, epidermal collarettes, and alopecia. Occasionally, the cat grooms excessively which can be interpreted as pruritus. Lesions around the nail beds and nipples are common.
6. **IF OVER 10 years of age at Onset:** Paraneoplastic pruritus is a rare disorder but can be observed in older cats with certain tumors. Pruritus associated with systemic and cutaneous tumors is more common in aged cats. Too often, the diagnosis is only made after prolonged attempts to identify more common causes of pruritus.

Diagnostics: Perform appropriate diagnostics based on prioritized differential list.

Trichogram	The microscopic examination of the hair (both the root and tip) may provide evidence of pruritus (fractured hair tips) or dermatophytosis (frayed root end).
	Many cats are reluctant to groom in front of the pet owner; therefore, owners may not be aware of the pruritus. Fractured hair tips can provide crucial evidence to confirm pruritus and convince the owner to proceed with diagnostic testing and treatment.
Fungal culture	Dermatophytosis is rarely pruritic; however, cats with miliary lesions may groom excessively.
Flea Combing	This should be one of the first diagnostic tests employed.
	The identification of fleas or flea dirt will confirm this common cause of feline skin disease.
	The flea comb material may also be used for microscopic identification of other ectoparasites (Cheyletiella, ticks, and other mites)
Cytology	The identification of bacterial (folliculitis), yeast, or acantholytic cells (pemphigus) will help guide additional diagnostics and treatments.
	Eosinophils are commonly found regardless of the primary etiology.
Skin Scrapes	The identification of Demodex (common), Cheyletiella, and Notoedres mites (uncommon) would confirm these diagnoses.
Fecal	Examination will occasionally reveal ectoparasites (mites) that were not found

floatation	on routine skin scrapings.
Flea Control Trial	Capstar administered every other day for 1 months effectively prevents flea feeding and the pets exposure to flea saliva. Aggressive Flea control on all pets in the home. Often, cats will have no evidence of flea infestation but respond to aggressive flea control.
Therapeutic trial for Demodex	Since skin scrapes for <i>Demodex gatoi</i> can be falsely negative in infected cats, a therapeutic trial consisting of 6 weeks of lime sulfur dips may be needed to confirm or rule out this differential.
Skin biopsy	This is often the quickest way to collect the most information regarding a dermatosis. Even if not diagnostic, enough information can be gathered to guide additional diagnostic tests or therapeutic trials (allergic dermatitis compared to folliculitis compared to paraneoplastic dermatitis).
Food Trial	Currently, a dietary food trial is the only way to confirm or eliminate food allergy dermatitis as a cause of pruritus. There are no in vitro testing methodologies that correlate with clinical disease.
	The most allergen restricted yet palatable diets should be used for a 10-week trial.
	Home-prepared diets provide better allergen restriction and are often more palatable than commercial diets, but are not balanced or complete.
	Following the 10-week elimination food trial, a dietary challenge should be used to confirm the diagnosis of food allergy. Often other treatments are initialed during the 10-week period. A food challenge will confirm or eliminate the restricted diet as the cause of improvement and thus substantiate the diagnosis of food allergy.

Summary:

Since flea allergy is the most common cause of skin lesions in cats, it is imperative to rule out flea allergy dermatitis through an aggressive flea control trial. The key to success is a thorough dermatologic workup. By considering and ruling out the more common or more easily treatable causes of feline dermatitis, success is easily achievable with out the need for steroids.

HOW THINGS WORK: PAIN PATHWAYS, RECEPTORS, DRUG TARGETS

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In the last 10 years, the veterinary profession has undergone what can only be described as a sea change in perspectives about animal pain and pain control. In many ways the issue of pain management in animals closely parallels that in human pediatrics, whereby the patient is non-verbal and the clinician must rely on personal/staff observations and the reports of the patient's advocate (in some ways this parallel extends to human geriatrics, whereby the patients may be once again non-verbal and a caregiver is the patient's advocate). Thus it is that physicians have also long struggled with the critique of under-managing pain in children^{1,2} the cognitively impaired,³ and the elderly.^{4,5}

Under- (or un-) managed pain elicits a cascade of debilitating neuro-hormonal effects that result in hypertension, catabolism, immunosuppression, and in what can be a terminal event, bacterial translocation and sepsis. This is called the "stress response." With under- (or un-) managed pain, patients at best recover more slowly from their condition, and at worst, may develop severe, even life-threatening complications.

However, the effect is not limited to pain of an acute nature. In addition to discomfort and physical disability, the capacity of chronic pain to impair cognition is becoming increasingly recognized in humans. A global summary of statistically significant findings in 42 studies of patients with chronic musculoskeletal pain revealed that deficits of memory, attention, psychomotor speed, and mental flexibility all can be attributed as a consequence of chronic pain, independent of other causes.⁶ In animals, for all of these reasons, under-attended, under-managed pain can become a criterion for euthanasia.

Pain itself is normal, and when physiologic it is protective. But undermanaged pain, as it becomes extended in time and intensity, becomes maladaptive and debilitating. And the younger the patient, the more long-term consequences of undermanaged pain because of the enhanced plasticity of the spinal cord: hypersensitivity to thermal stimuli can be documented years after the initial sets of painful experiences in both animals and humans.⁷ Thus for clinicians in a veterinary practice, their staff, and their clients, the first step to developing an aggressive, integrative pain management system is to internalize how dangerous and damaging undermanaged pain is to their patients. In fact, until so convinced, stocking drugs on a shelf and writing down protocols stands little chance of successful hospital-wide implementation.

The neuro-anatomic, physiologic, and molecular basis of nociception is a rapidly evolving field of study. Once-simple models are now understood to be highly complex and supremely inter-related sets of dynamics. The "Gate Control Theory", offered in 1965 by Melzak and Wall, proposes a feedback mechanism that controls activation of pain fibers by allowing or inhibiting impulses through the "gate."⁸ Nothing that we now understand about nociception challenges the basic operational premise of the Gate Theory. What is new and growing is the illumination of its details.

Nociceptors are specialized nerve fibers that have their dendritic endings in peripheral tissue, with several different subtypes identified. These nerve fibers have receptors that respond to mechanical and chemical stimuli but may be polymodal for touch, pressure, heat, cold, itch, and other sensations. When activated by the appropriate stimulus, a signal is said to be *transduced*, and the nerve endings depolarize. The signal is then conducted, or *transmitted*, electrobiochemically in an afferent direction, that is, towards the spinal cord. There, in the dorsal horn, the signal is *modulated*, that is either enhanced or dampened. Synapses are made with secondary neurons which ascend up the spinothalamic tract of the spinal cord to the thalamus, where another synapse occurs with tertiary neurons, which then project to the cerebral cortex where *perception* occurs. However, in addition to these ascending pathways to the brain are descending, inhibitory pathways; and under the proper conditions conduction can occur from the spinal cord down the peripheral nerve fibers in an anti-dromic fashion to the site of original transduction.

The fastest of the nerve fibers are the small but fully-myelinated A-beta sensory fibers which involve the sensations of touch, pressure, vibration, and proprioception. Somewhat slower are the thinly-myelinated A-delta fibers which stem from mechano-, thermo-, and nociceptors involved in sharp physiologic and acute pain. C-fibers are large and unmyelinated and hence very slow conductors of mechanoreceptors and nociceptors involved in dull, aching chronic pain. From somatic sites the cell bodies of these nerve fibers are located in the dorsal root ganglia, and from visceral sites, the sympathetic ganglia. The terminal endings of these fibers are highly tropic in the dorsal horn, with somatic A-delta and C fibers occurring in the most dorso-lateral aspect (Laminae I and II), somatic A-beta fibers terminating in the deeper Laminae II, IV, and V, and visceral A-delta and C fibers scattered throughout each of these Laminae.⁹ However, the tropism, inter-connectivity, and even phenotype of these various neurons is not static, rather the dorsal horn can exhibit dramatic plasticity, changing and altering form and function depending on a wide variety of factors: age (the younger the more plasticity), type and duration of stimulus, gender (or sexual status i.e. presence or absence of androgenic hormones), and others.

At the peripheral site of transduction, stimulus comes in the form of heat (transient vanilloid receptor 1, TRPV1), cold (cold- and menthol receptor 1, CMR1), membrane distortion, or cell damage releasing fatty acids and free ions from cell membranes. Each of these stimuli open non-specific cation channels on the peripheral endings of A-delta and C-fibers, which allows an inward Na⁺, K⁺, or Ca⁺ current. When a critical threshold of intracellular Na⁺ and/or Ca⁺ is reached, then activation and opening of voltage-gated cation channels occurs, which propagates depolarization afferently along the nerve fiber membrane.¹⁰ In addition, the free fatty acids are catalyzed by phospholipase-A2 into arachadonic acid, providing the substrate for cyclo-oxygenase metabolism and the initiation of the inflammatory cascade through a number of mediators e.g. prostaglandins, H⁺ ions, cholecystikinin, histamines, Substance P, bradykinins, leukotrienes, and many more,¹¹ all highly noxious stimuli that bind to their own receptors on the nociceptor nerve ending, exacerbating or continuing the cation influx. The peripheral nerve fiber transmits its signal to the spinal cord, terminating in the dorsal horn.

In the dorsal horn, the nociceptors terminate and release various highly bioactive molecules across synapses to interneurons (also called *second-order* neurons). Chief among many of these in the classic model is the excitatory amino acid glutamate, which binds to AMPA receptors on the interneuron. This binding causes a sodium/potassium channel to open, allowing Na⁺ to flow freely through the cell membrane into cytoplasm (and K⁺ out into the extracellular space), which

elicits an action potential: the interneuron depolarizes and the impulse is transmitted afferently to the brain. However, as quickly as it opens, an AMPA receptor will close, unless the stimulus is sustained. If the stimulus is in fact sustained, not only will the AMPA receptor remain open, but the accumulation of intracellular Na⁺, will phosphorylate adjacent NMDA receptors, releasing a magnesium “plug.” The NMDA receptor is now open and free to allow calcium to inflow into the neuron, further depolarizing it for an extended period of time.¹² NMDA activation is now well-established in its role of potentiating hypersensitization and neuropathic pain.¹³

The second-order, or projection neurons, upon which the peripheral A- and C-fibers synapse, are characterized as wide dynamic range (WDR, sensitive to a variety of sensations, including pain) and nociceptive-specific (NS, pain-only) neurons. They ascend the spino-thalamic tract to terminate in the thalamus, with projections (via third-order neurons) to the reticular, limbic, homeostatic-control, and cortical somatosensory regions of the brain¹⁴. Here the spatial and temporal qualities of pain become more than an unpleasant sensation, but transcends to a physical and emotional experience as well.

Inhibitory neurons, some intraspinal and some descending from the brain, synapse on the second-order neurons as well. Here the neurotransmitters are inhibitory in nature and include gamma amino butyric acid (GABA), norepinephrine (NE), certain serotonin (5-HT₃), B-endorphin, and others¹⁵. Furthermore, circulating endogenous opioids bind to kappa and delta (less so mu) receptors (closing Ca⁺ channels, and opening K⁺ channels, respectively), hyperpolarizing the cell. A basal level of interconnectivity occurs between afferent A-beta, A-delta, C-fibers, interneurons, and intra- and descending inhibitory neurons.¹⁶ Lastly, the supporting glial cells (astrocytes, microglia, oligodendrocytes) in the spinal cord, whose purpose was once thought to be merely structural in nature (providing synaptic architecture, host defense, and myelin, respectively), are now thought to be highly integrated into the pain process, particularly with regards to chronic pain.¹⁷ Recently described is the tetrapartite synapse, which includes an astrocyte, microglial cell, and pre- and post-synaptic neuronal terminal.¹⁸ A recently isolated chemokine, fractalkine, appears to be a neuron-glia cell signal, activating glially-dependent pain facilitation (in a recent rat model, blocking the one known fractalkine receptor in rats diminished the development of neuropathic pain).¹⁹ Indeed, the glia may play a primary role with regards to synaptic strength, plasticity, and sensitization in the spinal cord, which does exhibit substantial change under the influence of chronic pain.²⁰

Sustained nociception begins to alter the dynamic considerably, and pain can quickly move from its physiologic, protective nature to a maladaptive one. The constant presence of inflammatory and bioactive mediators at a peripheral site forms a “sensitizing soup” that creates a constant barrage of excitatory neurotransmitters in the dorsal horn. The opening of the calcium channel begins a cascade of events that in some cases becomes irreversible. An influx of calcium ion causes activation of Protein Kinase C (PKC), which in turn elicits production of nitrous oxide (NO), which then diffuses back across the synapse and through the terminal ending of the afferent nociceptor. This causes K⁺ channels to close and also the production of Substance P, a profoundly excitatory bioactive molecule, which then flows back across the synapse once more to bind on neurokinin (NK-1) receptors of the interneuron²¹ (expression of the NK-1 receptor appears to also contribute to opioid-induced hyperalgesia and tolerance²²). Not only does the interneuron stay depolarized, but a phenotypic change may be induced where it may not reset. Expression of *c-fos*, *c-jun*, and *Knox-24* genes transcribe new (probably aberrant) proteins that produce permanent microstructural changes of the neuron that result in reduced firing threshold,

upregulation of central neuronal activity, downregulation of inhibitory activity, expansion of the receptive field, peripheral hypersensitivity and intensified pain responses to further stimulation.²³

Furthermore, the afferent nociceptor will conduct a signal efferently, in an anti-dromic fashion. There, at the peripheral site of original stimulus, it releases Substance P and calcitonin gene-related peptide (CGRP), another highly bioactive excitatory compound, which elicits further release of inflammatory mediators and recruiting and activating other previously innocent-bystanding nociceptors, further bombarding the dorsal horn with impulses.²⁴ As the feedback loop persists, more and more cells express *c-fos* and other genes, Nerve Growth Factor is stimulated into production (suspected to be from glial cells), and more interconnections are made between types and locations of neurons in the spinal cord.²⁵ These interconnections are not isolated to somatosensory neurons, for they have been shown to newly express adrenoceptors which are activated by catecholamines. Sympathetic stimulation may then result in nociception,^{26, 27} and may in fact be central to the pathophysiology of neuropathic pain. Moreover, neuropathic pain is associated with alterations in receptor location (more places on more axons) and sensitivity to excitatory amino acids (greater) throughout the nervous system.²⁸ Eventually, when the process of pain is located centrally (in the spinal cord) rather than at the site of the original stimulus, the pain is said to be “neuropathic” in origin. Once neural pathways are thus sensitized, the physiologic (and physical) responses to pain may persist, even when the peripheral nerves themselves are blocked (or even transected).²⁹ Clearly, at this point, pain has become a disease unto itself.

Summary of terminology used to describe this sensitized state:

Peripheral hypersensitization: generation of an ever-present “sensitizing soup” of inflammatory mediators (prostaglandins, bradykinin, cytokines, neuropeptides), activation of quiescent (silent/sleeping) bystander nociceptors from non-injured tissue, reduction of threshold in normally-high threshold nociceptors.

Central hypersensitization: increase in the excitability of neurons in dorsal horn of spinal cord, cumulative depolarization (“wind up”) amplifying the neuronal activity in dorsal horn, generation of Nerve Growth Factor which promotes interconnections between formerly segregated types and locations of neurons, expression of new receptors, and phenotypic modification of nerve function.

Neuropathic pain: the extension of hypersensitization which is the initiation of transmitting a pain impulse (spontaneous depolarization) in the absence of noxious stimuli, or out of proportion to it.

In both acute and chronic pain, other non-neural peripheral tissues are not exempt from physical changes as well. Reflex muscular spasms are not only themselves painful, they may compromise vascular supply, and the resulting ischemia can result in release hydrogen ions and ATP, which are also highly sensitizing agents. This can result in altered, maladaptive conformation and gait, leading to abnormal stresses on ligament, tendon, cartilage, as well as and hyperirritable bands of contracted muscle (myofascial trigger points, TrP).³⁰

Glial cells (astrocytes, microglia, oligodendrocytes) in the spinal cord, whose purpose was once thought to be merely structural and macrophage-like in nature (providing synaptic architecture, host defense, and myelin, respectively), are now thought to be highly integrated into the pain process, particularly with regards to chronic pain.³¹ Recently described is the tetrapartite synapse, which includes an astrocyte, microglial cell, and pre- and post-synaptic neuronal

terminal.³² A recently isolated chemokine, fractalkine, appears to be a neuron-glia cell signal, activating glially-dependent pain facilitation (in a recent rat model, blocking the one known fractalkine receptor in rats diminished the development of neuropathic pain).³³ Indeed, the glia may play a primary role with regards to synaptic strength, plasticity, and sensitization in the spinal cord, which does exhibit substantial change under the influence of chronic pain.³⁴

There is no one moment when pain is transformed from physiologic to “acute” to “chronic” to “hyperesthetic” to “allodynic” to “neuropathic”. Rather it exists on a continuum with a high degree of biologic variation from patient to patient. There is also recent evidence that anxiety in the acute setting, mediated by cholecystikinin rather than mobilization of the hypothalamic-pituitary-adrenal axis, plays a major role in creating a chronic, hyperalgesic state.³⁵

Historically, the focus of analgesia has been to diminish transduction (e.g. local anesthesia, anti-inflammatories) and perception (e.g. opioids), and indeed these remain crucial components of a multi modal approach to pain management. The most exciting area of attention today however is in the dorsal horn, by enhancement of inhibitory modulation of nociception and interrupting the feedback loop that results in exaggerated pain responses and perception. As greater understandings emerge of the molecular and physiologic bases of pain emerges, new opportunities for intervention also emerge.³⁶

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RE-THINKING NSAID: NEW CONCEPTS WISEST, HIGHEST, SAFEST USE

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It is clear that non-steroidal anti-inflammatory drugs will likely remain the most commonly utilized modality to manage pain. They are highly effective, commonly available, licensed for use in dogs, and generally quite safe...and because inflammation is one of the underlying physiologic mechanisms by which pain is generated, their mechanism of action puts this class of drug as among the most important tools in the toolbox. At the same time, however, like other classes of drugs, NSAIDs do carry the potential for adverse effects that might range from the mild to the catastrophic.

The primary mode of action is to inhibit cyclooxygenase 2 (COX2), the enzyme that is expressed at site of inflammation and results in the production of pro-inflammatory and vasoactive prostaglandins. Also, through poorly understood mechanisms, likely by modulating multiple gene expression pathways,¹ it may inhibit central perception of pain. Several superior products are now labeled for use in dogs (and some in cats), making them among the most popular of pain management medications in veterinary medicine. All seem to be effective, and head to head studies now emerging may help to reveal objective differences if they are present. The main limitation of all NSAID's revolves around the potential for adverse effects, since both COX 1 and COX 2 enzymes may be constitutive, that is, consistently present and crucial to the production of cyto-protective prostaglandins (COX1 especially in the GI tract and renal tubules, COX2 in the renal tubules). Thus the primary adverse effects of non-selective NSAID's may include GI erosion/ulceration and nephrotoxicity. COX1-sparing NSAIDS should have a dramatically diminished GI toxicity profile, but will maintain their risk for nephrotoxicity. Rarely and on an idiosyncratic basis, hepatotoxicity may occur.

The GI and renal adverse effects can be expected to occur most commonly in higher risk patients, e.g.: hypovolemia, hypotension (including anesthetic procedures especially those not supported by intravenous fluids), pre-existing GI or renal disease, overusage, and the inappropriate combination with other NSAID's or corticosteroids. Notable in this last category is client use of aspirin in their pets, which may be unbeknownst to the clinician unless specifically queried in a thorough history. Unique to aspirin, this NSAID produces a cyto-protective lipoxin through the COX pathway;² thus when COX is inhibited through the use of another, concurrently-given NSAID, the potential for GI toxicity is considerably enhanced. The relative roles and molecular dynamics of COX1, COX2, and a possible new variant COX3, is still being elucidated and the "final word" on the optimal COX-selective or -sparing effect in order to maximize effectiveness and to limit toxicity, is yet to be heard. Acetaminophen may elicit some of its analgesic effects by inhibiting the COX3 variant, and recent studies suggest that it may also inhibit COX2-mediated production of PGE2.³

Lipoxygenase also metabolizes arachadonic acid, but instead of prostaglandins the byproducts are leukotrienes, which are potent attractors of PMN's and promote their adherence to endothelium. One commercial veterinary NSAID, tepoxalin, inhibits LOX as well as balanced

COX enzymes. In any use of NSAID's, the potential for adverse effects needs to be made clear to pet owners, and for any extended use, regular metabolic monitoring should be performed.

The long-term use of NSAID's may increase the chances of adverse effects, in particular GI and nephrotoxicity, but the evidence is beginning to suggest that whether a patient experiences an AE is less dependent on longevity of use than it is on biologic predisposition to occur, and improper use. One study looking at 19 cases of NSAID-induced GI perforation in dogs established that >90% of these patients were given concurrent corticosteroids or another NSAID (or no washout period between two different NSAID's), or were given higher than recommended doses for extended periods of time.⁴ To wit, the American Gastroenterological Association reports that 44% of respondents in a survey reported personally using higher than the recommended doses of NSAID,⁵ and up to 40% of pet owners may give their pets' aspirin at one time or another. Thus the veterinary clinician must be strident in their query of pet owners about other medications in the pet's history, and in their instructions about chronic usage. In general, a 5-day washout between NSAID's is recommended, and 10 days specifically for aspirin. Strict monitoring of clinical status and renal and hepatic values becomes a vital part of long-term NSAID usage.

NSAID's have been used, although cautiously, in patients with stable chronic renal failure; a rule of thumb is to multiply the dosing interval by the factor of the serum creatinine to account for decreased renal clearance (for example if it is a Q 24 hour medication and the creatinine is 3.0 mg/dl, then the modified interval would be $24 \times 3 =$ every 72 hours or once every 3 days).⁶ Due to the reno-protective effect (vasodilation) of COX2-mediated metabolites of arachadonic acid, it is possible that patients with documented CRD would be candidates to receive more balanced NSAID's.⁷ Patients receiving an ACE-I for protein-losing nephropathy or cardiovascular disease are at risk for adverse effects; in this case, the NSAID may blunt the effect of the ACE-I, and the ACE-I can potentiate the risk renal damage. Close monitoring of all of these patients, using the least effective doses, is warranted, and they are possibly more suited for balanced COX inhibitors.⁸ Where possible, the use of other modalities may allow lower NSAID doses which may in turn increase the safety profile, although there is no data to support this construct.

In all cases of NSAID use, the practice must consistently and reproducibly educate clients regarding the potential adverse effects of this class of drug. More than $\frac{3}{4}$ of individuals reporting adverse NSAID events to the FDA hotline feel that their veterinarian did not inform them adequately of possible side effects, and/or failed to give the client the drug information sheets provided by the pharmaceutical company.⁹

Acetaminophen appears to have weak COX-1 and COX-2 inhibition, but may inhibit a centrally-expressed COX-3 and a partial COX1 (PCOX-1) enzymes, mediating an analgesic effect by dulling the pain sensory system.¹⁰ Acetaminophen is contraindicated in cats and in patients with liver disease, and should be used with caution in dogs due to limited experience and diminished metabolism when compared to humans.¹¹

Tips For NSAID Use:

In the surgical setting, whether to administer NSAID pre- or post-operatively is a clinical decision. The edge in efficacy goes to pre-operative use in both humans¹² and dogs.¹³ Level 1 evidence evidence suggest safety of pre-operative NSAID in humans,¹⁴ as do multiple RCT's

(Level 2 evidence) in healthy dogs vis-à-vis renal function even with moderate intra-operative hypotension.^{15, 16, 17, 18, 19} However, it is axiomatic that patients undergoing general anesthesia should have the benefit of intravenous fluid support, which would further increase the safety margin of NSAID use even pre-operatively. One unpublished case series of cats undergoing de-sexing not receiving IVF did result in a number of patients experiencing acute renal failure with only 1 or 2 doses of various NSAID.²⁰ Customarily, NSAID are given during the expected course of post-operative inflammation, as little as 3 days for minor or elective procedures, up to several weeks for more major surgery.

Minimizing the Risks of NSAID: 8 Easy Steps

1. Complete medication history, including specific queries re: aspirin, other NSAIDs by brand name, “steroids”, “cortisone”, nutritional supplements (some have COX-inhibiting mechanisms, some may actually contain aspirin itself). Other medications:
 - a. Highly protein-bound drugs e.g. phenobarbital, digoxin, cyclosporine, chemotherapy agents
 - b. furosemide and ACE-inhibitors
 - c. Potentially nephrotoxic drugs e.g. aminoglycosides, cisplatin
2. Patient selection – caution or avoid NSAID with existing or anticipated
 - a. Low flow states such as hypotension, hypovolemia, dehydration (and all anesthetic procedures should have IV fluid support, blood pressure monitoring), and CHF
 - b. renal, cardiac, or hepatic dysfunction
3. Verbal and written client instructions to avoid the medications describe in #1 above, and to discontinue and alert the hospital at the first sign of an AE (see #4)
4. Recognize the earliest signs of AE and withdraw immediately: most often decreased appetite or an episode of vomiting, usually within 2-4 weeks but can occur at any time
5. Laboratory monitoring, frequency depending on the risk factor of the patient
 - a. Ideally within first month of initiating, then Q 6 months thereafter in low-risk patients
 - b. For higher risk patients, frequency Q 2 – 4 months
6. Use NSAID-sparing strategies (multi-modal approach to pain management) to find the lowest effective dose over time, if the patient requires extended use
7. Washout aspirin, meloxicam (10 days), other NSAIDs, or corticosteroids (3 days) if possible prior to initiating NSAID
8. Gastroprotectants if necessary (to treat suspected gastropathy or to prevent in the event no washout can occur); proton-pump inhibitors are superior to H2 antagonists²¹, and misoprostol (Cytotec®, a PGE2 analogue) is considered the drug of choice in humans although sucralfate (Carafate®) can also be helpful

Summary of literature findings,^{22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33} including Systematic Reviews on veterinary NSAIDs,³⁴ with specific regards to toxicities^{35, 36} and treatment of osteoarthritis in dogs³⁷:

Hepatotoxicity: Rare: 1.4 cases per 10,000 dogs (0.052%), usually between 2-4 weeks of initiating; elevated liver enzymes not a risk factor. “FDA ADE reports suggest that hepatic

toxicity can occur with any veterinary NSAID, and there are no reports identifying a particular NSAID as having an increased risk of idiosyncratic hepatic toxicity in dogs.” (KuKanich 2012) “No extensive studies (properly designed i.e. crossover, randomized) of clinical dogs using all available veterinary NSAIDs; but existing studies suggestive that the COX-1 sparing NSAIDs produce a lower frequency of GI lesions” (Kukanich 2012); while at the same time “...studies suggest that the more highly COX2-selective inhibitors may actually produce more adverse effects when underlying gastric damage is present.” (Goodman 2009)

Gastrointestinal toxicity

- #1 clinical sign associated w GI toxicity: vomiting, followed by inappetance (Stanton 1989, Lascelles 2005, Neiger 2003) ; however it is possible for erosions and ulcers to be silent and occur prior any prior clinical signs. (Stanton 1989, Wooten 2010)
- #1 risk factors for GI perforations are excessive dose and concurrent with other NSAIDs or corticosteroids, or combinations of these. (Lascelles 2005)
- Signs of GI toxicity usually emerge within 2-4 weeks but can occur at any at any point during administration. (Hampshire 2004, Robertson 2008)
- Veterinarians are generally very poor at communicating NSAID toxicity risk factors to pet owners or even handing out client information sheets with NSAID prescriptions. (Hampshire 2004).
- 22,200 NSAID incidents reported to the ASPCA Animal Poison Control Center between 2005-10; the most common drugs cited was ibuprofen, followed by aspirin, naproxen, whether accidental ingestion or owners administering the drug.
- With veterinary NSAID’s, “No study has comprehensively compared the AE profile or efficacy of the currently produced NSAIDs in head to head trials. Similarly, none of the studies have produced consistent results to indicate any of the veterinary- approved NSAIDs as being associated with more or less adverse effects in clinical patients.” (KuKanich 2012)
- Pre-operative administration in dogs is superior in efficacy to post-operative. (Lascelles, 1998), consistent with results of multiple studies performed in humans
- Nephrotoxicity: Administered pre-anesthetically in healthy dogs with controlled modest hypotension, no adverse affect on renal function was detected by the various outcomes measured such as serum BUN and creatinine concentrations, urine GGT:creatinine ratio, urinalysis, and glomerular filtration rate via scintigraphy. (Ko 2000, Bostrom 2002, Lobetti 2000, Crandell 2004, Bostrom 2006). “However, these are much less sensitive indicators than renal blood flow, altered distribution of blood flow through renal cortex, urine sodium clearance (which have not been extensively studied).” (KuKanich 2012). The renal safety of NSAID in healthy patients undergoing anesthesia is also established in humans.³⁸ Similar studies have not been in cats (undergoing anesthesia), but one feline study revealed no alteration in glomerular filtration rate, as measured by iohexol clearance, after 5 days of oral meloxicam.³⁹
- Tissue healing: highly COX-2 selective NSAIDs have demonstrated delayed bone healing in rabbit and rodent models, and 1 study in dogs demonstrated delayed TPLO fracture repair with long-term use of NSAID.⁴⁰ However, normal tissue healing is rapidly restored when the NSAID is withdrawn;⁴¹ a meta-analysis in humans reported no increased risk of nonunion with NSAID exposure when only the highest-quality studies were assessed;⁴² and of deracoxib, carprofen, and firocoxib, 299 dogs received one of

these NSAIDs in the FDA approval process and none reported to have delayed fracture healing or nonunion fractures.⁴³ Note: A systematic review in humans undergoing intestinal resection/anastomosis does reveal an increased risk of leakage at the anastomotic site with the use of COX-2 selective NSAIDs,⁴⁴ but this effect has not been studied in animals.

- Veterinary NSAID's studied for chronic use (28 days to 1 year) demonstrated satisfactory safety profiles in dogs, with discontinuation of use due to adverse effects in the 3-5% range (Innes 2010). While a similar systematic review has not been performed in cats, the 6-month safety of reduced-dose meloxicam (0.01-0.03 mg/kg/day) has been reported,⁴⁵ and the results of one retrospective study suggested that a long-term maintenance low-dose of 0.02 mg/kg/day of meloxicam can be safely administered to cats older than 7 years even if they have chronic kidney disease, provided their overall clinical status is stable (and may actually slow the progression of CKD in some cats).⁴⁶ A new feline COX-2 selective NSAID, robenacoxib, revealed no toxicities at 6X and 10X labeled daily dose for the duration of the 6-week study.⁴⁷
- Under outcomes measured, carprofen may demonstrate chondroprotective effects in dogs.^{48, 49, 50, 51} Studies of other veterinary NSAIDs, and in cats, are still needed.
- In two studies of long-term use of NSAID for canine osteoarthritis, clinical scores continued to improve during the entire duration of the study. (6 months carprofen, Autefage 2010⁵²; 1 year firocoxib, Autefage 2012⁵³)
- Therefore with regards to long-term use of veterinary NSAIDs in the treatment of canine osteoarthritis, "... a number of lines of evidence suggest the potential theoretical benefits of continuous versus intermittent NSAID analgesic therapy for osteoarthritis." (Innes 2010)

New in NSAID's

- In 2015, the FDA designated a new "piprant" class of "non-COX-inhibiting NSAID." It represents a new targeted inhibition of strictly the EP4 receptor of PGE2, with the hope of a prospectively wider safety margin than historical COX-inhibiting NSAID. The first drug in this class, grapiprant, is expected to be approved in the U.S. in 2016. Safety data reveals minimal ADE at 15x expected labeled dose administered for 9 months.⁵⁴
- robenacoxib (Onsior®)¹: available in Europe, and approved by the FDA for post-operative pain in cats in the U.S. in 2011, this unique COX-2 selective^{55, 56} NSAID has a short plasma half-life in this species (1.7 H), yet accumulates in inflammatory exudates for up to 24 hours.^{57, 58} This novel "tissue-specific" character appears to fulfill the promise to provide both safety and efficacy in cats, and in fact, while approved only for 3 days post-operatively, a 6-week trial in cats revealed the drug to be well-tolerated even at 3X and 5X the labeled dose.⁵⁹ In a recent study of cats undergoing ovariohysterectomy, robenacoxib appeared to elicit superior post-operative pain control than meloxicam.⁶⁰ Note: robenacoxib has been approved for use in dogs in Europe with a similar profile as found in cats, but is not imminently available in the U.S.
- RevitaCAM™ (Abbott Animal Health), an oral transmucosal meloxicam spray, has been approved in the UK for acute and chronic musculoskeletal pain in dogs and cats, and may be in the pipeline for U.S. release as well. It is a 5 mg/ml solution, with the dosage being 0.2 mg/kg for the first dose followed by 0.1 mg/kg/d thereafter; the specially-

designed dispensary glass bottle (3 different concentrations sizes) dispenses a metered dose/spray per pump. The U.S. formulation of this product, OroCAM™, was withdrawn from the market in 2013.

- mavacoxib (Trocoxil®, Europe only) - sustained-release NSAID approved for chronic pain in dogs. A 2010 search for literature yielded an abstract on the pharmacokinetics of the drug² but no information on efficacy; the manufacturer reports 30 days after 1 dose.⁶¹
- nitronaproxen (Naproxcinod®, Europe only for humans) is a cyclooxygenase-inhibiting nitric oxide-donating drug (CINOD) in Phase III trials that appears to have the analgesic efficacy of the parent NSAID but with a greatly reduced incidence of negative side effects because of the positive effects of the NO.⁸ Naproxcinod has been shown to control the pain of osteoarthritis in humans.⁶²

¹ Xiao-Min W et al Rofecoxib modulates multiple gene expression pathways in a clinical model of acute inflammatory pain, *Pain* 128(1-2) March 2007: 136-147

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³ Arndt J¹, Claudia Giesen,, Lee Y-S, Kim H, et al. Acetaminophen selectively suppresses peripheral prostaglandin E2 release and increases COX-2 gene expression in a clinical model of acute inflammation. *Pain* 2007 129(3):279-286

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⁷ Gaynor, JS, Clinician's Update, NSAID's: Liver & Kidney Disease in the Osteoarthritis Patient Dec 2006:2-4

⁸ Atkins, CE, Clinician's Update, Canine Heart Disease and NSAID's, June 2007:2-4

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Re-Thinking Opioids: New Concepts, Technologies, Formulations

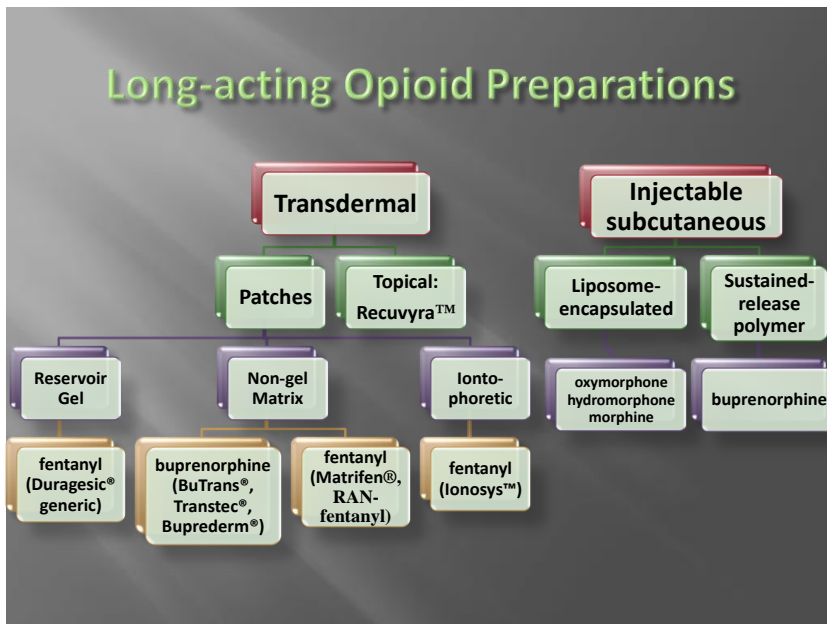
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A limiting factor of many analgesic medications, including opioids, is a relatively short duration of action. Extending opioid duration of action to last several days provides several unique therapeutic advantages:

1. Eliminates plasma concentration peak/troughs encountered with repeated parenteral dosing.
2. Steady state of opioid can be achieved at plasma levels sufficient to elicit analgesia without the use of intravenous constant rate infusion or transdermal patches.
3. Eliminates multiple injections to administer opioid periodically throughout at 24-hour period.
4. Eliminates staffing requirements to reliably administer an opioid through a 24-hour period, further eliminating compliance and variability issues. (This feature in laboratory animals provides, by extension, consistency for the study model).

Thus there is an increasing interest in sustained-release and/or long-acting parenteral opioid formulations and technologies in humans and animals, several of which have been investigated: some adopted from human use, some from compounding pharmacies, and one of which has recently received FDA approval in dogs. The applications are to be found both in clinical patients and in research animals (where the stress of handling to administer B-QID drugs, and 24-hour staffing requirements to do so, is considered confounding to study models).

A number of different technologies have been explored and utilized to achieve long-acting opioid effect:



In animals, the efficacy, durability, and tolerability of liposome-encapsulated (LE) hydromorphone has been demonstrated in dogs, with adequate serum levels up to 4 days¹ and superior analgesic effect 12 hours post-ovariohysterectomy compared to subcutaneous morphine.² This same formulation, route, and dose demonstrated favorable pharmacokinetics and tolerability in rhesus macaques when compared to subcutaneous or intravenous hydromorphone.³ Similar studies with LE oxymorphone and hydromorphone have been performed in laboratory animals demonstrating durability, tolerability, and effectiveness (rhesus macaques⁴ and rodents^{5, 6, 7}). However liposomes are highly labile, with limited shelf-life and expensive to produce. Currently, no commercial LE-opioid product is available on the market.

Fentanyl is a short-acting opioid conventionally administered via constant-rate intravenous infusion. It has also been available in the U.S. as a transdermal patch formulation since 2005 (Duragesic®), labeled in humans for breakthrough cancer pain, and has been studied (and used off-label) in dogs, cats, and rabbits for post-operative and other kinds of pain. Study results have demonstrated utility in these species^{8, 9} but also wide variability in serum concentrations, even when application of the patch is standardized.^{10, 11, 12} Add the variables of patch location (trunk, vs. limb, with different dermatologic characteristics and temperature), body condition (amount of adipose tissue in skin), and even how well and properly the patch stays secured to the skin (or not), and the reproducibility of reliable plasma levels from patient to patient should be called well into question. An additional concern with this technology is human exposure to the reservoir, which is not only easily divertible for illicit use, but also potentially dangerous including death when ingested (and to which children are particularly susceptible; 10 fatal cases have been reported since 1997). Indeed, one study examining the residual fentanyl in the patch after attachment to dogs for 72 hours concluded that it was significant and could easily lead to human intoxication¹³ (note: the study did not analyze remnant fentanyl actually located on the dogs' skin, but it might be presumed that exposure to humans could also occur via skin-to-skin contact). While Duragesic patches maintain some traction in veterinary medicine, and may be particularly indicated for

specific patients, the plasma variability, and prospective liability, has caused it to carry less popularity in veterinary medicine than it once did.

Fentanyl patches also come in a non-reservoir, gel matrix patch (Matrifen® and generics), indicated for chronic pain. The fentanyl in these products is more difficult to divert for illicit use, but the liability issue remains similar to Duragesic® and insofar as the author is aware, no PK or PD studies on this type of patch have been published in animals.

More recently, a long-acting fentanyl product with a novel delivery system was approved in dogs for post-operative pain (Recuvyra®, Elanco). This product makes use of patented Metered Dose Transdermal Spray (MDTS) technology (Medistend™, already in human use to deliver estrogen and testosterone supplementation, contraception, and other compounds) and is labeled to provide plasma levels of fentanyl adequate to provide analgesia for 4 days. The solution is highly concentrated fentanyl, 50 mg/ml, and is applied at a dose of 1.2 mg/lb (2.7 mg/kg) to healthy skin at the dorsal scapular region 2-4 hours before surgery; staff are advised to wear personal protective equipment and the patient is to wait a minimum of 2 minutes before returned to a holding area; the area of application should not be touched for 5 minutes. During this time, a penetration enhancer (octyl salicylate, commonly used in sunscreen) drives the fentanyl into the stratum corneum, 15 layers of dead cells in a lipid matrix. A depot of fentanyl resides within the lipid matrix and it slowly releases into circulation by passive diffusion along a concentration gradient.

Several peer-reviewed articles have explored the pharmacokinetic and pharmacodynamic properties of Recuvyra™.^{14, 15, 16, 17, 18} Analgesic plasma levels of fentanyl (1 ng/ml) are reached within 3 hours, with a C_{max} of 13 hours. Interestingly, the drug has demonstrated “Flip flop” kinetics: the rate of absorption is slower than the rate of elimination.

Skin to skin contact does not lead to human exposure, in fact the administration model appears not to work in human skin. However, skin to mucous membrane exposure (i.e. touching the area and then touching to mouth, eyes, etc.) is possible. Therefore it is advised that the application area not be touched for 72 hours, and to segregate the patient from children during that time. The adverse effects are reported to be those typical of any opioid, and reversible if necessary with the opioid antagonist naloxone 0.04 mg/kg IM; however due to the short duration of naloxone and the long-duration of the drug, sequential re-administration will be required, every 1-3 H.¹⁹ Further, naloxone at customary doses can be expected to reverse both the adverse and analgesic effects; recall however that low-dose naloxone (0.004 mg/kg) has been shown to reverse the adverse effect, while maintaining the analgesic effect, of opioids;²⁰ however such use of naloxone has not been evaluated with Recuvyra™. Off-label, nalmefene may antagonize the fentanyl for longer than naloxone (extrapolating from human doses, 0.25 µg/kg - 30 µg/kg; an oral formulation of nalmefene is commercially available, used for alcohol addiction in humans, but no data exists in dogs).

Buprenorphine is recently available for humans as a transdermal patch (Transtec®, BuTrans®, Buprederm®). Rabbits and rodents achieved rapid plasma levels (1-24 hours) with peak analgesic activity with the tail-flick and writhing model at 3-4 hours and sustained for 72 hours of the study.²¹ However in one feline study using a 35 mcg/h patch, plasma levels were negligible and there were no changes in thermal thresholds.²² The experience in dogs is somewhat better. In one canine study utilizing a 70 mcg/h patch resulted in sustained plasma concentrations of 0.7-1.8 ng/ml within 36 hours of application.²³ Another canine study utilizing a 52.5 mcg/h found peak plasma levels of 1.54 ng/ml and analgesic efficacy to be non-inferior to IV buprenorphine in mechanical & thermal thresholds within 36 hours of application and lasting until the patch was removed; however there was some inconsistency as 3 of the 10 dogs recorded negligible plasma levels.²⁴ An additional clinical canine study found the 70 mcg/kg patch to be non-inferior to SC buprenorphine post-ovariohysterectomy.²⁵

Buprenorphine is also available in a compounded (non-FDA approved) sustained-release formulation (Zoopharm) administered subcutaneously. The technology utilizes a biodegradable polymer which is dissolved in a biocompatible solvent. Upon SC injection, the polymer precipitates or coagulates upon contact with aqueous body fluid to form an implant matrix, from which the active drug is gradually release as it degrades. Several such biodegradable polymers are in common commercial use, such as N-Methyl-2-Pyrrolidone (NMP, which degrades via the tricarboxylic acid cycle into CO₂ and water), and Triactin (TCN, glycerin and acetic acid). These polymers are in common use with cosmetics, various medical beads and implants, and several other products familiar to veterinarians including doxycycline-impregnated pericuteic treatments and absorbable sutures.

Unpublished PK data in dogs report plasma levels adequate for analgesia for over 72 hours,²⁶ but there are anecdotal reports of prolonged and in some cases dramatic sedation especially at the higher end of the dosage range (0.27 mg/kg SC) in larger dogs.²⁷ Unpublished PK data in cats superior maintenance of plasma levels adequate for analgesia over 3 days when compared to repeated OTM dosing.^{28, 29} One published PD study in cats found SR buprenorphine to be non-inferior to Q 12 H OTM dosing for three days post-ovariohysterectomy, with minimal adverse effects when administered at 0.12 mg/kg SC.³⁰ Similar positive outcomes have been observed in unpublished studies with non-human primates³¹ and rodents,³² and in one published rat study.³³

In July 2014, a new veterinary formulation of buprenorphine was FDA-approved in the United States and introduced into the marketplace (Simbadol®, Abbott). At 1.8 mg/ml it is 6 X more concentrated than the human commercial product Buprenex® (0.3 mg/ml). It is labeled for post-surgical pain in cats, a 24-hours duration with one injection at 0.24 mcg/kg SC; it can be given daily for up to 3 days. The labeled dose is 0.24 mg/kg, approximately 10x higher previously recommended. The operating premise is that the ceiling effect of buprenorphine limits adverse effects while allowing the extended analgesic duration. Each 10 ml vial allows for approximately 15 doses. The shelf-life of an unopened vial is 21 months, and once opened, 28 days.

Long-acting opioids remain an area of active research, and new products may be finding their way into the marketplace, and possibly FDA-approved, in the coming years.

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THOSE “OTHER” PAIN DRUGS: FACT & FICTION

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Outside the realm of NSAID and opioid exist a broad range of medications that exert an analgesic effect, or otherwise modify and protect against pain, by manipulating various targets along the nociceptive pathway.

ALPHA-2 AGONIST

Alpha-2 and opioid receptors are co-located on central nociceptors, and use of the two drug classes together is highly synergistic for sedation and analgesia. Alpha-2 binding pre-synaptically reduces NE production is reduced and sedation occurs; binding post-synaptically, analgesia is produced. It also blocks NE receptors on blood vessels, resulting in vasoconstriction; the resulting hypertension parasymphatically induces bradycardia, which is extended by a subsequent direct decrease in sympathetic tone although central perfusion is maintained. It has a versatile dosing profile where low (5 mcg/kg) and even micro-doses (0.25 – 1 mcg/kg, resulting in volumes as low as 0.01-0.03 ml even in larger dogs) with opioids are clinically useful and minimize the cardiovascular effects. Be mindful that these lower doses will shorten the duration of the drug, and the analgesic effects may wane prior to the sedative effects. The safety and pain modifying effect of dexmedetomidine constant rate infusions have been described.¹

KETAMINE

A phencyclidine dissociative anesthetic ketamine exerts a pain-modifying effect predominantly as potent NMDA-receptor antagonist. Ketamine binds to its receptor inside the NMDA receptor, i.e. the calcium channel would already have to be open and active for ketamine to exert its effect. However, once bound, it decreases the channel's opening time and frequency, thus reducing Ca⁺ ion influx and dampening secondary intracellular signaling cascades. Hence it is unlikely (and has not been shown) to be truly analgesic in nature. Rather, subanesthetic ketamine constant rate infusion (CRI) has been shown convincingly in humans to have pain-preventive, anti-hyperalgesic, anti-allodynic effects^{2,3,4,5,6,7} and existing studies in the dog appear support a similar clinical effect in dogs^{8,9,10} (not yet studied in a feline surgical model). The International Veterinary Academy of Pain Management has adopted a position that the pain-modifying effects and safety warrant the consideration of subanesthetic ketamine as part of a multi-modal approach to transoperative pain management, especially in patients with risk factors that may dispose them to exaggerated or maladaptive pain states.

INTRAVENOUS LIDOCAINE

The mechanisms behind a pain-modifying effect of systemic lidocaine remain an area of investigation but appear to include its ability to enter the nociceptor cell body in the dorsal root ganglion. In humans the evidence is strong for safety and the beneficial effects of intravenous lidocaine (IVL) on pain after abdominal surgery in humans (although not other surgeries eliciting somatic pain)¹¹ and possibly horses, including both

pain and return of bowel function. Systemic, intravenous infusion of lidocaine has also been shown to elicit a sustained effect on neuropathic pain in humans,¹² although this has not been studied in companion animals. It is anesthetic sparing in dogs and cats, but current evidence for a pain-modifying effect in these species remains inconclusive.^{13, 14, 15, 16, 17, 18} IVL can still be suggested as a safe and sparing adjunct to opioid and other analgesics for abdominal surgery, trauma, and pancreatitis at a dose of 50 mcg/kg/min, in dogs; and this has been used for 24 – 48 hours.

Some investigators discourage the use of IVL in cats due to negative cardiovascular effects,¹⁹ but anecdotally has been utilized in clinical practice. Systemic, intravenous infusion of lidocaine has also been shown to elicit a sustained effect on neuropathic pain in humans,²⁰ and may have a specific point of action in the brain.²¹

Note: Alpha-2 agonists, systemic lidocaine, and ketamine CRI are sedating and can be profoundly anesthetic-sparing especially in combination with opioids.¹ If administered during anesthesia, induction doses and maintenance vaporizers should be adjusted downward accordingly, and can often be at 1% or less. The drug concentrations and fluid rates may be adjusted to fit the needs of the individual patients. Rate calculators are available on www.vin.com (Library/Calculators) and www.vasg.org/resources_&_support_material.

TRAMADOL

In humans tramadol is known to exert its pain-modifying effect through two metabolites; one enhances inhibitory neurotransmitters (serotonin, norepinephrine), and the other (O-desmethytramadol, or “M1”) metabolite is a weak opioid (1/100th the mu-receptor affinity of morphine). However, tramadol has a very short half-life (1.7 hours) in the dog,²² and it appears that dogs produce very little of the M1 opioid metabolite.^{23, 24, 25} Nevertheless pharmacodynamic studies in dogs have demonstrated the anesthetic-sparing^{26, 27} and pain-modifying effect of *parenteral* tramadol,^{28, 29, 30, 31, 32, 33} Convincing evidence for a pain-modifying effect of *oral* tramadol remains elusive at this time.³⁴ Furthermore, PK studies in dogs reveal that not only are plasma levels much lower following oral administration than in humans, sequential dosing for several days leads to dramatic reductions of those plasma levels^{35, 36} (suggesting saturation of GI active transport sites, increased metabolism, more robust first-pass effect, or a combination). One study of oral tramadol reports a statistically significant increase of mechanical threshold levels, but only at the 5- and 6- hour time point.³⁷ One study does find oral tramadol effective as *part* of a multi-modal analgesic protocol to control cancer pain,³⁸ but others have found it (not unsurprisingly) inferior as a solo agent to multi-modal analgesic approaches to control post-operative pain.^{39, 40} The short half-life of the drug suggests up to a Q-6 hour treatment regime, but one unpublished abstract on the effectiveness of tramadol administered once daily in canine osteoarthritis appears encouraging.⁴¹ In contradistinction to dogs, cats do produce the mu-agonist M1 metabolite,⁴² and a pain-modifying effect has been demonstrated in both a thermal threshold⁴³ and clinical surgical model,⁴⁴ as well as a case series of use of oral tramadol

in a flavored compounded form⁴⁵ (the drug is otherwise quite bitter). Toxicity and safety data are lacking in both dogs and cats.

GABAPENTIN

Gabapentin is an anti-convulsant that analgesic properties predominantly by down-regulating voltage-dependent calcium channels⁴⁶ but other mechanisms probably exist as well (while structurally similar to GABA, it is not a direct agonist, although it may have indirect effects on GABA metabolism such as increasing intracellular stores). Because of its effectiveness and tolerability, it is in widespread use for humans with neuropathic and other maladaptive pain conditions,^{47, 48, 49, 50, 51} and this suggests, along with published case reports,^{52, 53, 54} a strong rationale for the utilization of gabapentin in analogous conditions experienced by dogs and cats. The utility of gabapentin for osteoarthritis is demonstrable in rodent models,^{55, 56} one canine study suggests a disease-modifying effect in experimental osteoarthritis,⁵⁷ but no clinical studies have been published investigating gabapentin canine OA. However, case reports exist of successful use in treating non-OA neuropathic pain conditions in both dogs^{58, 59, 60} and cats.⁶¹ In cats, one unpublished study is reported to demonstrate a benefit of gabapentin in naturally-occurring osteoarthritis,⁶² in addition to a case series of chronic musculoskeletal pain.⁶³

Systematic reviews in humans support safety and benefit of transoperative oral gabapentin for post-surgical pain.^{64, 65, 66, 67, 68} The evidence in dogs and cats for efficacy in acute pain currently is disappointing,^{69, 70, 71, 72} but one case series utilizes gabapentin in cats with acute traumatic musculoskeletal injuries.⁷³ Pharmacokinetic studies in dogs reveal that it may have a half-life of 3-4 hours in the dog⁷⁴, suggesting a TID administration schedule although anecdotally BID appears to be useful. The primary adverse effect in dogs appears to be somnolence (as in humans) which usually will spontaneously resolve over a few days acclimation time. For chronic pain dosing, a general consensus is that doses are initiated at 3 mg/kg and gradually tapered upwards as the patient can tolerate to a target dose range of 20 mg/kg. In the perioperative setting, dose based on the experience in humans is 10 mg/kg.

AMANTADINE

Amantadine exerts a pain-modifying effect as an NMDA receptor antagonist⁷⁵ and remains a research focus for chronic pain (but not specifically osteoarthritis) in humans⁷⁶ One study does demonstrate utility as an adjunct to NSAID in dogs with refractory osteoarthritis.⁷⁷ Toxicity and kinetic studies have been performed in humans⁷⁸ and cats⁷⁹ but not in dogs. Amantadine dosing range is 3-5 mg/kg orally once daily..

TRICYCLIC ANTI-DEPRESSANTS

TCA's exert their analgesic activity by enhancing synaptic norepinephrine and serotonin (inhibitory transmitters) in the dorsal horn of the spinal cord, although it has other effects including anti-histamine, anti-cholinergic, NMDA receptor antagonism, and sodium channel blockade. It has a balanced NE and serotonin effect, and thus is among the more sedating, anti-cholinergic, and effective of various TCA's.⁸⁰ As a class, TCA's are the most effective medications for neuropathic pain in humans.⁸¹ However in dogs there exists only a single case report of utilizing amitriptyline for neuropathic musculoskeletal

pain, and in cats the experience in idiopathic cystitis⁸² (also now termed “Pandora Syndrome” for its description as a somatic pain syndrome). In humans TCA’s can have an unfavorable side effect profile which limit their use for neuropathic pain despite their efficacy. Customary doses of amitriptyline are 1-2 mg/kg BID in the cat and dog, but a recent review article suggests 3-4 mg/kg based on its PK profile in these species.⁸³

SS(N)RI’s

These compounds exert their effect by increasing serotonin +/- norepinephrine in the synaptic cleft. At least one SSNRI, duloxetine, has a chronic pain label in humans, but bioavailability is poor in dogs⁸⁴ and clinical efficacy is lacking.

Note: many drugs and compounds enhance monoamines and/or serotonin and caution should be undertaken when or if used in combination. Examples include: tramadol, TCA’s including amitriptyline and clomipramine, SS(N)RI’s, amantadine, metoclopropamide, selegiline, amitraz, mirtazepine

ACETAMINOPHEN

Acetaminophen is contraindicated in cats. In dogs, several older studies reveal a pain-modifying effect in orthopedic surgery,^{85, 86} and pharmacokinetic data has been reported.⁸⁷ The literature does not appear to support that the dog has any special proclivity towards hepatotoxicity.

MAROPITANT

Maropitant is a central antiemetic through blockade of Substance-P to the NK-1 receptor, which is also involved in pain processing. The true pain-modifying effect in dogs remains uncertain despite one study in dogs revealing an anesthetic-sparing effect⁸⁸ and another a non-inferior effect to morphine in an ovariohysterectomy model.⁸⁹

BISPHOSPHONATES

Administered by IV infusion, the class of drug exerts anti-osteoclast activity and can contribute to pain relief in dogs with bone cancer.⁹⁰

CORTICOSTEROIDS

Corticosteroids are not analgesic in and of themselves, but likely exerts an indirect pain-modifying effect through by reducing inflammation. Its utility as an analgesic therapy in dogs and cats has not been reported, although several studies of intra-articular corticosteroids suggest possible chondroprotection.^{91, 92, 93, 94}

POLYSULFATED GLYCOSAMINOGLYCANS

Parenterally-administered PSGAG products have regulatory approval as safe and effective chondroprotectants, supported by independent studies.^{95, 96, 97}

NEUTRACEUTICALS AND OTHER ORAL SUPPLEMENTS

Oral nutritional supplements represent a wide spectrum of compounds as single agents or in combinations. Evidence for a pain-modifying effect of these various products remains mixed at this time although some display some encouraging data may exist for some. If

neutraceuticals or herbal supplements are made part of a treatment plan, the Task Force suggests mindfulness towards: quality control, potential drug interactions with other medications (for example, some OTC joint products contain aspirin), and ingredients derived from endangered species.

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**2015 AAHA/AAFP PAIN MANAGEMENT GUIDELINES:
WHAT IT MEANS FOR YOUR PRACTICE**

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The robust advances in pain management for companion animals underlie the decision of AAHA and AAFP to expand on the information provided in the 2007 AAHA/AAFP Pain Management Guidelines for Dogs and Cats. The 2015 Guidelines can be found at these URL's:

(https://www.aaha.org/public_documents/professional/guidelines/2015_aaha_aafp_pain_management_guidelines_for_dogs_and_cats.pdf and <http://jfm.sagepub.com/content/17/3/251.full.pdf+html>)

The Guidelines continue the trend in all branches of medicine toward evidence-based consensus statements that address key issues in clinical practice. Although not a review article, the Guidelines represent a force multiplier for the busy practitioner, consolidating in a single place current recommendations and insights from experts in pain management. The recommendations of the guidelines Task Force are evidence based insofar as possible and otherwise represent a consensus of expert opinion. These notes contain the key applied principals for veterinary clinicians.

TRANSOPERATIVE PAIN

Devising an evidence-based top-tier trans-operative pain management strategy is within the scope of any practice to achieve. The framework of effective pain management systems rests solidly on the foundation of recognition/assessment, pre-emption, and using multiple modalities. Multiple modalities allow for intervention at several different places of the nociceptive pathway, increasing effectiveness and minimizing the need for high or protracted doses of any one particular drug. It is well-established in human medicine, for example, that the use of adjunct medications will minimize the use of PCA (patient-controlled analgesia) opioids with a resultant decreased incidence of adverse effects such as nausea and constipation.^{1,2,3,4} In short, employing the modest use of multiple modalities is not only more effective, it avoids the over-reliance (higher doses, longer duration) of any *one* modality...thus limiting the likelihood of adverse effect from *any* modality.

The basic construct is a 4-legged stool:

ANXIOLYTICS

Anxiety contributes directly to the hyperalgesic state through cholecystikinin-mediated "nocebo" effect.⁵ A number of studies in humans support the idea that patients who are highly anxious or stressed pre-operatively experience higher pain scores post-operatively. These observations are also found in many animals studies, where restraint, social defeat, rotation – all things veterinary patients experience in the normal pre-surgical setting in

order to draw blood, place catheters, etc. – contribute to hyperalgesia.⁶ Thus the first leg of a strong transoperative pain management protocol does not involve the use of analgesics in and of themselves, but anxiolytics and not just pharmacologic ones i.e. low-stress handling techniques¹ and pheromones in addition to tranquilizers/sedatives. In this modality class, clinicians may choose between phenothiazines (e.g. acepromazine), benzodiazepines (midazolam or diazepam), or alpha2 agonists (dex/medetomidine).

OPIOIDS

Opioid receptors are distributed ubiquitously throughout the body and can be found in most central and peripheral tissues. Several opioid different receptor types and subtypes have been isolated, each with a variant effect; activation of an opioid receptor inhibits presynaptic release and postsynaptic response to excitatory neurotransmitters. The proposed mechanism includes opioid receptor coupling with the membrane-associated G protein; this leads to decreased intracellular formation of cAMP which diminishes calcium channel phosphorylation (closing off the channel) and opens potassium channels enhancing potassium influx. The resulting effect is hyperpolarization of the neuron and blockade of Substance P release. Nociceptive transmission is thus greatly impeded.⁷ Opioids in combination with anxiolytics discussed above can induce a profound sedating neuroleptanalgesic effect to the patient's benefit.

Similarly, a number of different opioid drugs are available which vary in their relative potency and receptor affinity, and a complete discussion of their similarities and differences are available in a number of resources. Briefly, however, of the pure mu agonists, morphine remains the prototype in widest use; it has no ceiling effect on analgesia or respiratory depression, elicits histamine release, and causes vomiting at low doses (higher doses, IV doses, and chronic use do not elicit vomiting, presumptively by interaction with mu receptors in the antiemetic center⁸). Cats lack glucuronate metabolism, resulting in minimal production of the analgesic M6G metabolite⁹, therefore morphine may not be the ideal opioid for use in this species. Oxymorphone (Numorphan®) and hydromorphone (Dilaudid®) do not elicit histamine release (therefore may be wiser choice in cases of hypovolemia e.g. trauma, dehydration), and nausea may be less pronounced, but they have a much shorter duration of action than morphine; also, hydromorphone in particular is implicated in episodes of hyperthermia in cats.¹⁰ Fentanyl in a transdermal patch (Duragesic®) remains useful in veterinary medicine though a number of studies have demonstrated wide kinetic variability in veterinary patients due to species, body condition score, body temperature, surgical procedure, where and how well the patch is placed, etc.^{11,12} A long-acting veterinary transdermal Fentanyl preparation, labeled for 4 days post-surgical pain relief in dogs, is now commercially available (Recuvyra™). Buprenorphine is a partial agonist on the mu receptor though it has greater affinity than morphine (and will displace it if given together). A great benefit of the drug in veterinary medicine is that its pKa (8.4) closely matches the pH of the feline oral mucosa (9.0), which allows for nearly complete absorption when given buccally in that species¹³, with kinetics nearly identical to IV and IM administration,¹⁴ and eliciting very little sedation. Simbadol® is a buprenorphine

¹ <http://drsophiayin.com/lowstress>, <http://www.catvets.com/guidelines/practice-guidelines/handling-guidelines>

FDA-approved product labeled for 24 hours of post-surgical analgesia in cats; a compounded sustained-release buprenorphine product purported to last for 3 days is also commercially available, although it is not FDA approved for safety and efficacy. Butorphanol is a mu agonist and a kappa antagonist; its very short duration of action in the dog (approx. 30-40 min) makes it a poor choice for an analgesic in this species, though used parenterally it has utility as an adjunct with other medications such as alpha-2 agonists. Tramadol (Ultram®), in contradistinction to humans, does not appear to have any opioid activity in the dog (although it does in cats).

Opioids for all their effectiveness may create clinical challenges as well. In the acute setting, opioid-induced dysphoria, hyperalgesia, and respiratory depression may be encountered; recognizing and having strategies for counteracting their signs will minimize the complications that they present.¹⁵

NSAID

The primary mode of action is to inhibit cyclooxygenase 2 (COX2), the enzyme that is expressed at site of inflammation and results in the production of pro-inflammatory and vasoactive prostaglandins. Also, through poorly understood mechanisms, likely by modulating multiple gene expression pathways,¹⁶ it may inhibit central perception of pain. Several superior products are now labeled for use in dogs (and some in cats), making them among the most popular of pain management medications in veterinary medicine. All seem to be effective, and head to head studies now emerging may help to reveal objective differences if they are present. The main limitation of all NSAID's revolves around the potential for adverse effects, since both COX 1 and COX 2 enzymes may be constitutive, that is, consistently present and crucial to the production of cyto-protective prostaglandins (COX1 especially in the GI tract and renal tubules, COX2 in the renal tubules). Thus the primary adverse effects of non-selective NSAID's may include GI erosion/ulceration and nephrotoxicity. COX1-sparing NSAIDS should have a dramatically diminished GI toxicity profile, but will maintain their risk for nephrotoxicity. Rarely and on an idiosyncratic basis, hepatotoxicity may occur. The GI and renal adverse effects can be expected to occur most commonly in higher risk patients, e.g.: hypovolemia, hypotension (including anesthetic procedures especially those not supported by intravenous fluids), pre-existing GI or renal disease, overusage, and the inappropriate combination with other NSAID's or corticosteroids. Notable in this last category is client use of aspirin in their pets, which may be unbeknownst to the clinician unless specifically queried in a thorough history. Unique to aspirin, this NSAID produces a cyto-protective lipoxin through the COX pathway;¹⁷ thus when COX is inhibited through the use of another, concurrently-given NSAID, the potential for GI toxicity is considerably enhanced. The relative roles and molecular dynamics of COX1, COX2, and a possible new variant COX3, is still being elucidated and the "final word" on the optimal COX-selective or -sparing effect in order to maximize effectiveness and to limit toxicity, is yet to be heard. Acetaminophen may elicit some of its analgesic effects by inhibiting the COX3 variant, and recent studies suggest that it may also inhibit COX2-mediated production of PGE2.¹⁸ Lipooxygenase also metabolizes arachadonic acid, but instead of prostaglandins the byproducts are leukotrienes, which are potent attractors of PMN's and promote their adherence to endothelium. A veterinary NSAID, tepoxalin (Zubrin®) that inhibits LOX as well as balanced COX enzymes is no longer

commercially available. In any use of NSAID's, the potential for adverse effects needs to be made clear to pet owners, and for any extended use, regular metabolic monitoring should be performed.

LOCOREGIONAL ANESTHESIA

Local anesthetics were once a mainstay of pain management in veterinary medicine, and may now be one of the most under-utilized modalities. Administered locally or regionally, they are the only modality that renders complete anesthesia to a site, i.e. no transmission of nociceptive impulses as long as the drug exerts its effect. . Initially used as a means of desensitizing tissues in order to “invade” tissues with scalpels; local anesthetics are enjoying a rebirth as powerful tools to prevent or reduce perioperative pain (as well as procedural and even chronic pain) and to reduce general anesthetic and concurrent analgesic (especially systemic opioid) requirements. There is no longer a reason to hold an “either-or” position; “for surgery either I use local anesthetics *or* I use general anesthesia”, in fact, there are many reasons to combine general and local anesthetic for surgical pain relief.¹⁹ A partial list of techniques, from the sublime to the more advanced include, topical/dermal/epidermal local anesthetics for IV catheter placement (e.g. EMLA®, LMX4®, or their generic equivalents), incisional blocks, infiltrative blocks, intra-peritoneal or intra-pleural blocks, perineural blocks (e.g. brachial plexus, and radial-ulnar-medial n. “Ring” block), intra-articular blocks, dental/orofacial n. blocks, epidurals, IV Regional Anesthesia (Bier) blocks, retrobulbar blocks, intercostals blocks, transdermal blocks e.g. EMLA® (see below) and Lidoderm®.

BEST OF THE REST

Cold Compression

Long known for its pain-modifying effect in humans, recent studies affirm a similar effect in dogs.²⁰

Alpha-2 agonist

Medetomidine and dexmedetomidine binds opioid-like receptors on C- and A-delta fibers, especially in the central nervous system. Binding pre-synaptically, NE production is reduced and sedation occurs; binding post-synaptically, analgesia is produced, and is profoundly synergistic with opioids. It also blocks NE receptors on blood vessels, resulting in vasoconstriction; the resulting hypertension parasymphatically induces bradycardia, which is extended by a subsequent direct decrease in sympathetic tone. However, central perfusion is maintained and the author has found a wide use for these alpha-2 agonists in acute and peri-operative setting, though only in combination with opioids and at doses much lower than suggested by the manufacturer. One particularly novel and user-friendly utility is IV micro-doses intra- and post-operatively, 0.25 – 1.0 mcg/kg. This may result in intravenous volumes of only 0.01 – 0.03 ml in even the largest of dogs.

Ketamine CRI

A phencyclidine dissociative anesthetic, the evidence is building for its pre-emptive and preventive effects when given at subanesthetic doses in an intravenous constant rate

infusion. Ketamine binds to a phencyclidine receptor inside the NMDA receptor, i.e. the calcium channel would already have to be open and active for ketamine to exert its effect. However, once bound, it decreases the channel's opening time and frequency, thus reducing Ca⁺ ion influx and dampening secondary intracellular signaling cascades. Hence it is unlikely (and has not been shown) to be truly analgesic in nature. Rather, it appears to be protective against hyperalgesia and central hypersensitization in the post-operative setting,²¹ including in the dog.²² Ideal sub-anesthetic ketamine plasma concentrations – eliciting the most benefit with the least adverse effect – has been reported at 2-3 mcg/ml, which can be achieved by administering ketamine IV CRI at 10 ml/kg/min.²³ This can be accomplished by placing 60 mg (0.6 ml of 100 mg/ml stock) ketamine in 1 L of fluids and administered at customary intra-operative rates of 10 ml/kg/hr. Post-operatively, the rate can be reduced to customary maintenance rates of 2 ml/kg/hr, which administers the ketamine CRI at 2 mcg/kg/min. A loading dose of 0.25 – 0.5 mg/kg ketamine IV is recommended prior to the initiation of the CRI in order to rapidly achieve plasma levels.

Adjunctive drugs: tramadol, gabapentin

In humans, tramadol (Ultram®) is described as a synthetic opioid with 1/100th of the affinity for the mu receptor as morphine but a much better analgesic effect than this would predict. This is likely due to the combined effect of a highly active M1 metabolite and serotonin- and norepinephrine (inhibitory neurotransmitters) agonism. However, recent work demonstrates that it appears to have a very short half-life (1.7 hours) in the dog,²⁴ and it appears that dogs produce very little of the M1 opioid metabolite.²⁵ Only recently have some studies demonstrated the probable clinical usefulness of *parenteral* tramadol in dogs,^{26, 27, 28, 29} but none so far with the oral formulation. The unfavorable PK profile of oral tramadol in dogs, and the lack of confirmatory data about its pain-modifying effect, should lend skepticism about its use as an analgesic in this species. Any pain-modifying effect of tramadol is likely derived from its serotonin- and norepinephrine-enhancing activity, therefore tramadol should only cautiously be used with other serotonergic medications such as tricyclic antidepressants.

Gabapentin is labeled for use as an anti-convulsant drug but is in widespread human use for its analgesic properties. While structurally similar to GABA, it is not a direct agonist, although it may have indirect effects on GABA metabolism such as increasing intracellular stores. Another leading hypothesis is that it exerts effect through interaction with the alpha-2-delta subunit of the voltage gated calcium channel.³⁰ Its utility in chronic, neuropathic pain states is well-established in humans,³¹ but more recently its utility in the transoperative setting is supported by a number of systematic reviews.^{32, 33, 34, 35, 36, 37} Pharmacokinetic studies in dogs reveal that it may have a half-life of 3-4 hours in the dog³⁸, suggesting a TID administration schedule. Starting doses are recommended in the 3-10 mg/kg range. The primary adverse effect in dogs appears to be somnolence (as in humans) which usually will spontaneously resolve over a few days acclimation time, but this AE not been a frequent occurrence in the author's experience.

CHRONIC PAIN

Several “Disability” indexes and “Quality of Life” scoring systems are available and can be used to semi-quantify patient comfort, mobility, and abilities, e.g.

CODI: Cincinnati Orthopedic Disability Index

HRQL: Health Related Quality of Life

Degenerative Joint Disease & Osteoarthritis

OVERVIEW: Arguably the most commonly-recognized chronic-pain condition in dogs and cats, DJD also presents some of the greatest challenges to treatment because of its inevitably progressive pathology, and difficulty in early recognition. DJD is the inclusive term that includes OA. Interestingly, cats appear to have a much higher incidence than dogs, with reports of 60-90% of all cats, including young ones, affected with radiographic DJD changes (which implies it may have a different pathophysiology than the DJD encountered in dogs). Complicating matters is that only in recent years has the question been asked (in humans and rodent models): does DJD lead to a neuropathic component? The answer seems to be yes, at least in some patients (for example, in 25% of humans with stifle OA patients). Indeed it might be safely surmised that DJD & OA specifically eventually does create a *maladaptive* – even if not abjectly neuropathic - pain state in most patients. That is, the perceived pain is disproportionately greater than would be expected by the extent of pathology alone.

DJD is typically envisioned of as a disease of bone and cartilage. And of course, physical examination – or even just movement - often will easily elicit the clicks, pops, and thunks attributable to osteophytes and bone-on-bone crepitation. But it is instructive to point out that the pain is not felt at the articular surfaces or what is left of them. Rather, the pain is felt in the peri-articular structures, from an inflamed synovium, when tension is placed on a fibrotic joint capsule, and when patients are asked to exert (even if just by standing or walking) weakened ligaments, tendons, and muscle. Thus OA is a disease of the entire joint organ, including dramatic synovitis, fibrosis, and atrophy...and the result is not just pain but progressive disability. Treatment has to be targeted accordingly.

As difficult a job exists to devise an EBM-approach to surgical pain, the challenge is many-fold times that with a multi-modal plan for osteoarthritis, which progresses in such variable ways, and with such variable responses to different interventions from patient to patient. Add in a paucity of properly-designed literature in dogs and cats vis-à-vis treatment, and widely divergent client (and veterinarian) values, and a real difficulty exists to formulate a standard approach.

However, it is possible to point out where the literature is strongest, and the neurophysiological/pharmacologic rationale the most compelling, and structure a plan around those modalities.

Weight optimization – clearly the #1 method for preventing DJD and a prime, however challenging, method for treating DJD. The role of adipose tissue as a mediator of systemic inflammation, the contribution of central obesity to chronic pain in humans

(doubling the risk for it³⁹), and the primacy of weight loss to diminish chronic pain signs and symptoms- is now a settled matter. In dogs with osteoarthritis, several studies illuminate the benefit of improving pain scores, mobility, and NSAID reduction with weight loss alone (even modest, i.e. only 5%⁴⁰). Indeed, it is probably not an overstatement to say that in an overweight patient, both the clinician and pet owner are wasting time and money on other interventions until and unless weight loss is achieved. NSAID – An abundance of literature in humans, dogs, and cats, as well as two Systematic Reviews of treatments for canine osteoarthritis therapy (which constitute Level 1 Evidence), reveal this class of drug to be, by far, the most predictably effective therapy. A number of studies now appear to point to the effectiveness as well as the safety of long-term NSAID in cats.^{41 42 43 44}

Parenteral polysulfated glycosaminoglycans (PSGAG), in particular Adequan® which is FDA-approved for the treatment of OA in dogs with bioavailability and efficacy supported by a number of independent studies. Another injectable glycosaminoglycan for horses, pentosan polysulfate (PPS, Cartrophen®), also has some evidence for benefit in canine OA⁴⁵ although not available in the U.S. The evidence for glucosamine and chondroitin in OA remains mixed at best, although some other ingredients of oral nutraceuticals such as avocado soybean unsaponifiables, MSM, green-lipped mussel, microlactin, and others offer suggestions for varying degrees of immunomodulating, chondroprotective, and pain-modifying effect.

Diet: EPA-rich diet in dogs,⁴⁶ DHA-rich diet in cats

Controlled Exercise: Long understood to elicit analgesia in humans, this non-pharmacologic modality is now considered equally important in dogs. The pain-modifying effect likely emerges from a convergence of benefits, including but not limited to: gate-theory-mediated analgesia, production of endogenous opioids, micostability of joint due to strengthened soft-tissue/peri-articular structures. Controlled exercise can be facilitated through a formal Physical Rehab program, and at-home programs can also be designed for the individual pet.

Other modalities, while having less robust evidence in the literature, are supported for use in the management of DJD

Acupuncture

Pain-modifying Analgesic Drugs (PMAD) especially gabapentin and amantadine, possibly Tricyclic Antidepressants (TCA) e.g. amitriptyline. Other drugs may find a role in the future e.g. Selective Serotonin Norepinephrine Reuptake inhibitors e.g. venlafaxine, and acetaminophen (+/- opioid e.g. codeine, hydrocodone) (DOGS ONLY).

Myofascial Trigger Point Therapy

Energy-based modalities e.g. Therapeutic Laser, Transcutaneous Neuromuscular Electric Stimulation (TNMES), Extracorporeal Shock-Wave Therapy (ESWT).

Biologic Therapy e.g. Mesenchymal Stem Cell transplantation, Platelet Rich Plasma.

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12 LOCOREGIONAL TECHNIQUES YOU SHOULD BE USING

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Local anesthetics were once a mainstay of pain management in veterinary medicine, and may now be one of the most under-utilized modalities. Administered locally or regionally, they are the only modality that renders complete anesthesia to a site, i.e. no transmission of nociceptive impulses as long as the drug exerts its effect. Initially used as a means of desensitizing tissues in order to “invade” tissues with scalpels; local anesthetics are enjoying a rebirth as powerful tools to prevent or reduce perioperative pain (as well as procedural and even chronic pain). There is no longer a reason to hold an “either-or” position; “for surgery either I use local anesthetics *or* I use general anesthesia”, in fact, there are many reasons to combine general and local anesthetic for surgical pain relief.¹

Local anesthetic drugs are extremely effective, inexpensive and easy to use. When local anesthetic drugs are administered, pain impulses originating in the periphery are blocked and prevented from reaching the central nervous system. This blockade has several positive consequences:

- The sensation of pain is alleviated or even eliminated for the duration of the block. Local anesthetic drugs work by blocking sodium channels in nerve membranes. Decreased permeability to sodium slows the rate of depolarization so that the threshold potential is not achieved and an action potential is not propagated, thus the pain impulse is not propagated. Local anesthetics bind more readily to ‘open’ channels, thus rapidly firing nerves are more susceptible to blockade.
- The likelihood that ‘wind-up’ or hypersensitization will occur is greatly decreased because the portion of the pain pathway called ‘transmission’ is blocked. Transmission involves the conductance of pain impulses from the peripheral nociceptors to the dorsal horn neurons in the spinal cord. The neurons in the dorsal horn are responsible for central sensitization. By blocking input to these neurons, central sensitization (or ‘wind up’) is less likely to occur.
- The analgesia allows the patient to be maintained under a lighter plane of anesthesia and this makes the anesthetic episode safer for the patient. In fact, local anesthetic drugs decrease the minimum alveolar concentration (MAC) of all anesthetic gases.
- Local anesthesia to a surgical site permits comfortable awakening from anesthesia, creating a sparing effect of other analgesic medications (thus decreasing the likelihood of their adverse drug effects)
- Local anesthetics have been associated with fewer exaggerated, sustained (i.e. maladaptive, neuropathic) pain states.
- Lastly, local anesthetics are recognized to have many beneficial effects beyond blocking nerve conduction; broad anti-inflammatory effects (reduced production of eicosanoids, thromboxane, leukotriene, histamine, and inflammatory cytokines; and scavenging of oxygen free radicals) and even antimicrobial, antifungal and antiviral effects.^{2,3}

Furthermore, local anesthetic blocks are extremely cost effective and can increase profits to the clinic.

Commonly used local anesthetic drugs in veterinary medicine include

- Lidocaine
 - Onset of action: rapid (less than 5 minutes)
 - Duration of action: 60-120 minutes
 - Dose 2-6 mg/kg (use the lower end of the dose in cats)
 - Convulsive dose in dogs: 11-20 mg/kg
 - Lethal dose in dogs: 16-28 mg/kg
 - 'Toxic dose' in cats reported as 6-10 mg/kg
 - The general recommendation for clinical use is ≤ 6 mg/kg in the dog and $\leq 3-4$ mg/kg in the cat.
- Bupivacaine
 - Onset of action: approximately 5-10 minutes after injection (up to 20 minutes)
 - Duration of action: 4 to 6 hours
 - Dose 1-2 –(4) mg/kg (use the lower end of the dose in cats)
 - Toxic dose in dogs: 5-11 mg/kg or potentially any amount given IV
 - Data is mostly anecdotal in the cat but the general feeling is that 3 mg/kg is the toxic dose.
 - The general recommendation for clinical use is ≤ 2 mg/kg in the dog and ≤ 1 mg/kg in the cat.

Adverse events caused by local anesthetic drugs: extremely rare but can include any of the following:

- Local tissue effects – swelling, bleeding, inflammation, 'tingling'? (unknown if this occurs in animals). A commonly held misconception is that local anesthetics impair wound healing – although they can powerfully inhibit the inflammatory component of cellular tissue influx, there is no evidence to support impaired wound healing. Both bupivacaine and ropivacaine have been implicated in myotoxicity, although it appears that this has not been listed as a complication in most human studies where these drugs were infused for 24 – 36 hours postoperatively into a wound bed. With proper technique and avoidance of needle induced trauma, local anesthetics can be used without the fear of negative effects on healing.
- Anaphylaxis – rare, more common with esters (but still rare)
- Central nervous system – muscle tremors, seizure, coma
 - At lower concentrations, depression of inhibitory neurons occurs and can cause cerebral excitation, which may lead to seizures. At higher concentrations, profound CNS depression with subsequent coma, respiratory arrest and death can occur. The latter is more likely following IV boluses of large doses.
- Cardiovascular system – the myocardial conduction system is sensitive to local anesthetics and IV boluses can result in cardiovascular collapse. **ONLY LIDOCAINE CAN BE ADMINISTERED IV (and never with epinephrine).**
- Methemoglobinemia – rare, but can occur in cats.

- Motor and autonomic nerves are also blocked by local anesthetics, and so motor weakness and vasodilation may occur with certain techniques. Blockade of essential nerve function, like that of phrenic nerve, or high epidural blocks, should be avoided. Motor weakness or paralysis of limbs, from spinal or major nerve trunk blockade is transient and as long as the patient is protected from injury and undue stress, should not be of consequence.

LOCOREGIONAL APPLICATIONS

The locality of administration is often limited only by the clinician's ability to learn various utilities and anatomic landmarks; few are outside the scope of any clinician to master. They include, but are not limited to local line or paraincisional blocks⁴, regional blocks such as carpal ring, dental nerve, and intercostal blocks, subcutaneous diffusion blocks, testicular blocks, intra-articular blocks, and epidurals. Facet blocks are commonly used in humans though not yet described in veterinary medicine, although recently a paravertebral block was described for dogs.⁵

Commonly used local anesthetic blocks in veterinary medicine

For many of the blocks listed below, a suggested volume of drug is listed based on the amount of drug that can physically be injected into the site. However, with all blocks, the total dose that the patient can receive should be calculated and the cumulative dose (add up the dose or volume injected for each block) should not exceed this total dose.

1. Cavity block

Pre-closure of celiotomy, the designated amount of local is injected into the abdominal cavity, or mixed into a final lavage without suctioning out. The safety (as long as no active venous bleeding) and efficacy of this simple technique has been demonstrated in multiple veterinary studies.^{6, 7, 8, 9} Intra-peritoneal infusion of LA has been utilized for painful abdominal conditions such as pancreatitis, bile and septic peritonitis. Intrathoracic injection of local anesthetic can be utilized for indwelling chest tubes, other painful intra-pleural conditions, and even to relieve the pain of pancreatitis (afferent innervation of the cranial peritoneum courses through the chest before entering the spinal cord).

2. Transdermal/cutaneous

Commercial transdermal products are extremely useful in facilitating catheter placement and for minor procedures involving the dermis and epidermis. A lidocaine/prilocaine ointment formulation (EMLA®, also comes as a generic) is placed on a shaved area and covered with a non-porous wrap (foil or cellophane). In humans it is recommended to have the product in place for 45 minutes to achieve full affect, but in the author's experience 15-20 minutes appears sufficient in dogs and cats. Penetration depth of analgesia has been reported to be time dependent and from 2-6 mm.¹⁰ Commercial 5% lidocaine patches (Lidoderm®) provides post-operative wound paraincisional analgesia.¹¹ However, Lidoderm® patches in fact are manufactured and labeled for post-herpetic neuralgia (Shingles), a very common form of chronic, neuropathic pain in humans. The pharmacokinetics of this product has been investigated in dogs and cats, with minimal systemic absorption noted.^{12, 13} The adhesive patches can be cut formed to the desired size and shape, for example on either side of an incision. One cautionary note is that an entire patch contains 700 mg of lidocaine, obviously a dose that would be toxic if ingested; therefore adequate

precautions need to be taken to ensure the patient is unable to access the patch. Studies in humans with moderate-severe stifle osteoarthritis reveal significant reduction in pain intensity after 2-week use of Lidoderm® patches¹⁴, and pain relief similar to that achieved by oral NSAID.¹⁵ Their potential for use in animals for chronic pain conditions (e.g. osteoarthritis, osteomyelitis, osteosarcoma) remains plausible but no applications are described in the veterinary literature.

3. 'Field' block

Blocking the 'field' of surgery. Local anesthetic drugs can be administered around the incision or directly into the incision. It is not true that lidocaine in an incision causes a delay in healing. Savvas et al (2008) reported that a subcutaneous incisional midline block prior to celiotomy provided superior pain relief, compared with postoperative bupivacaine or saline infiltration in dogs having a variety of abdominal surgeries.¹⁶ These authors used a dose of 2 mg/kg of 0.25% bupivacaine. Carpenter et al (2004) compared the effects of intraperitoneal bupivacaine with that of saline and lidocaine in dogs having ovariohysterectomy and found that the bupivacaine treated dogs received less supplemental analgesia and had improved pain scores.¹⁷ These authors used a higher than commonly recommended dose of bupivacaine (4.4 mg kg⁻¹ 0.75% bupivacaine diluted with saline to a volume of 0.88 ml/kg), in the cranial intraperitoneal space with an additional 2 ml of 0.75% bupivacaine on the incision prior to closure. Tobias et al 2006 did not find a benefit to a preoperative subcutaneous infiltration of 1.1 mg/kg bupivacaine in cats having ovariohysterectomy, but this dose is low compared to other studies.¹⁸ In another study in cats, lidocaine was infused subcutaneously, dripped on the ovarian pedicles and on the linea prior to closure, and the requirement for supplemental anesthetic doses of ketamine were significantly reduced, compared to cats treated with equivalent volumes of saline. The total dose of lidocaine used was 5 mg/kg. Results of veterinary studies suggest that incisional and intraperitoneal use of local anesthetics can spare opioid requirements and improve pain scores, and this is supported by multiple studies of similar techniques in humans having laparotomy.

4. Indwelling diffusion/wound catheter block (sort of a long term field block)

A relatively new strategy to extend the duration of local anesthesia may markedly help patients with moderate to severe surgical injury. Implantation of a catheter into the surgical wound site prior to closure allows repeated or continuous infusion of local anesthetics into the affected area. Indwelling, or 'soaker', catheters should be considered for large wounds or incisions that may be difficult to block or that may require continuous or intermittent delivery of drug for several days. The catheters can be buried in or near incisions and local anesthetic infused through the catheter to provide more long-term analgesia. Very useful for surgeries with large incisions, eg: amputations, mastectomies, etc. Local anesthetic drugs can be infused via a pump or administered by intermittent injection (eg, q 6-8 hour injections of bupivacaine). The catheter is generally removed in 48-96 hours. In humans, relatively costly FDA approved catheters^a are used. For veterinary use, two moderately priced types are commercially available^{b,c}. The basic form is a soft pliable catheter with tiny holes along the end that is implanted; functioning somewhat like a garden "soaker hose". Utility, efficacy, and safety has been demonstrated in humans (Liu et al, 2006) and dogs. reported.^{19, 20} Duration is commonly 2 days, and the veterinary utility includes: limb amputation, ear canal ablation, intercostal and sternal thoracotomy, celiotomy, and major soft tissue tumor excision, with excellent results and few complications.

5. **Oral blocks (Figure 1):** Blocks listed below will cause unilateral desensitization from the site of injection rostrally to midline.

Maxillary or infraorbital nerve block, cranial approach: The infraorbital nerve exits the infraorbital foramen, which can be palpated as a depression in the buccal mucosa dorsal to the distal root of the maxillary 3rd premolar (just cranial to the root of the 4th premolar or carnassial tooth in the area where the gingiva on the maxillary bone and the gingiva on the lip join together). Block the nerve by injecting local anesthetic under the gingiva just rostral to the foramen or insert the tip of the needle into the infraorbital canal and inject. Injecting into the foramen insures more caudal spread of the block but is not necessary if the oral surgery site is rostral to the foramen. Also, the foramen can be difficult to locate or to enter in small dogs and cats & infusion rostral to the canal is still useful as there will be some caudal migration of the local anesthetic into the canal. A vessel runs with this nerve so aspirate, then slowly infuse drug (0.1 to 1.0 ml).

Mandibular nerve block, extraoral approach: The mandibular foramen or the mandibular nerve can often be palpated on the lingual side of the mandible just rostral to the angle of the mandible and just caudal to the last molar in approximately the middle 1/3rd of the mandible (as measured from top to bottom). Regardless of whether or not the nerve or foramen can be palpated (often difficult to palpate in very small patients), the landmarks described above will be utilized for deposition of local anesthetic drug. The nerve ENTERS the mandible at the mandibular foramen and cannot be blocked between the mandibular foramen and the mental foramen. Landmarks are the same as those described above but the approach is from the outside, through the skin at the angle of the mandible. This technique is easier than the intraoral technique in cats and in some small dogs. Pass the needle through the skin along the medial aspect of the mandible with the needle perpendicular to the mandibular cortical bone, to the level of the foramen (again, aiming for a site just caudal to the last molar on the lingual side of the mandible). With a finger in the oral cavity the needle can be felt under the gingiva. When the site near the mandibular foramen is reached, aspirate and inject the local anesthetic drug (0.2-2.0 mls).

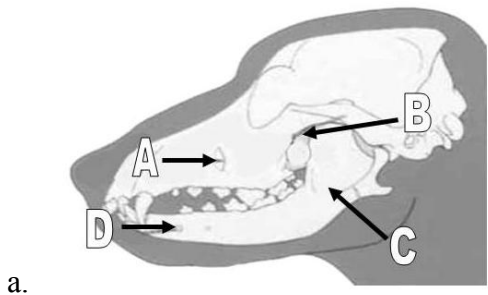


Fig 1 - Diagram of a dog's skull showing the locations of a variety of local anesthetic blocks. Cranial and caudal infraorbital block (A), inferior alveolar nerve or mandibular block (B), and mental nerve block (C). Landmarks for cats are the same as for dogs. Diagram used with permission from Pfizer Animal Health.

6. Testicular block

Isolate body of testicles, inject lidocaine or bupivacaine into the body of the testicle until you feel 'pressure', generally ½-2 ml per testicle in dogs and cats; the drug will migrate up spermatic cord. For incision directly over testicle, continue infiltrating as the needle exits the testicular body to block the skin and subcutaneous tissue. For incision in other location, inject local anesthetic in skin and subcutaneous tissue at site of incision.

7. Ovarian block

The mesovarium can be infiltrated with lidocaine, generally 0.5 mls per side in small dog up to 1-2 mls/side in large dog (up to 5 mg/kg total). Elevate ovary, infiltrate mesovarium, elevate

opposite ovary, infiltrate mesovarium, remove first ovary, remove the second ovary and proceed with the ovariohysterectomy.

8. Digit or paw block (Figure 2)

The three point (or four point) technique: Locate the carpus and the accessory carpal pad, inject 0.1-0.3 mls subcutaneously at three sites: 1) medial to the accessory carpal pad (blocks median nerve and palmar branch of the ulnar nerve); 2) lateral and proximal to the accessory carpal pad (blocks dorsal branch of the ulnar nerve); and 3) on the dorsal-medial portion of the carpus (blocks superficial branches of the radial nerve).

The Ring block: Similar to three point block but use a subcutaneous 'line' of local anesthetic all the way across the dorsum of the paw and another 'line' all the way across the ventrum of the paw to provide a 'ring' of local anesthesia that desensitizes the nerves described above.

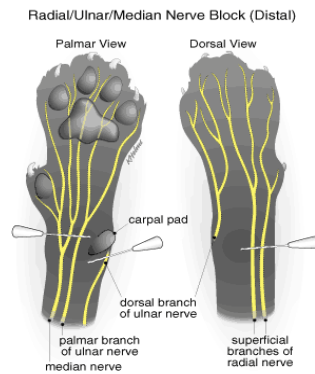


Fig 2 - Diagram of a cat's distal forelimb showing the locations for placement of local anesthesia for desensitization of the digits. Diagram used with permission from Tranquilli WJ, Grimm, KA, Lamont LA. [Pain Management for the Small Animal Practitioner](#). Teton New Media Jackson, WY, 2000.

9. Intercostal block

Inject local anesthetic in the tissues caudal to the proximal portion of the ribs. Inject local anesthetic in 2-3 rib spaces in front of and 2-3 rib spaces behind the area that needs to be desensitized.

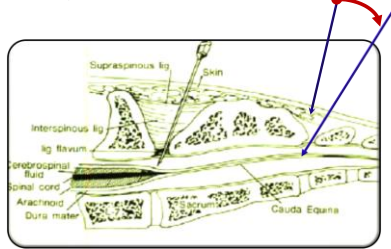
10. Brachial plexus block

Locate the point of the shoulder, the first rib and the transverse processes of the cervical vertebrae, insert a 2-3 inch needle (an epidural needle will work) at the point of the shoulder to the point where the tip of the needle is even with the first rib. Keep the needle horizontal during placement so that the tip does not enter the thoracic cavity. Aspirate, then inject 1/3 of the local anesthetic (2 mg/kg bupivacaine diluted with saline to a total 1 ml solution per 4.5 kg body weight) at this site, slowly withdraw the needle to the middle of the area to be blocked, aspirate and inject 1/3 of the local anesthetic. Withdraw the needle to a site just before it exits the skin, aspirate and inject the remaining 1/3 of the local anesthetic.

11. Sacro-coccygeal block²¹

Sacro-coccygeal

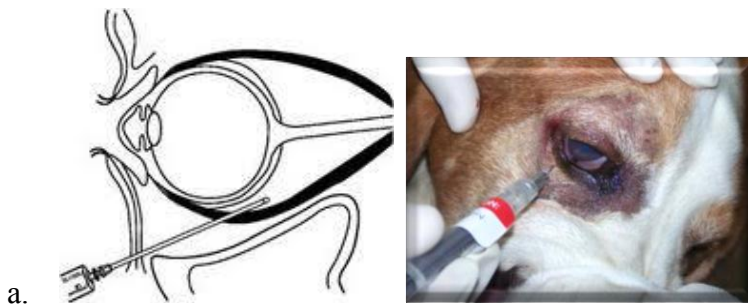
- Anal saccullectomy
- Perineal mass removal
- Other tail, perineal S'x
- Blocked cats
- PU



Indicated for perineal, tail procedures including: relief of urinary obstruction, perineal urethrostomy, anal saccullectomy, peri-anal mass removal, tail amputation. The patient is placed in sternal recumbency, palpate the space between the sacrum and the 1st coccygeal vertebra while dorsoflexing tail (between Cx 1-2 also acceptable). Clip & prep, Use a 25ga 1” needle to penetrate the skin at midline. Direct the needle at a 30 to 45 degree angle and continue through the interarcuate ligament. There may be a palpable “pop” when the ligament is penetrated; as the needle is advanced, there is no resistance upon entering the epidural space. If bone is encountered, keep the needle in the skin and slightly angle the needle cranially or caudally off the bone until the space is entered. The needle feels more firmly seated once the ligament is penetrated than it does in the subcutaneous tissues. Inject 0.5 ml of 2% lidocaine or 0.5% bupivacaine; there should be no resistance

12. Retrobulbar block^{22, 23}

With a 1 ½” 22g needle, enter suborbital above the lateral 1/3 aspect of the zygomatic arch. Direct caudo-medially to come up underneath and behind the globe. Aspirate and inject the local anesthetic.



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SUPPLEMENT THE ORTHOPEDIC CARE IN YOUR PRACTICE

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It should be acknowledged that owners use diets, supplements, herbs, massage, and acupuncture in their own health care and the expectation is for today's veterinarian is to have a basic knowledge of these complementary modalities for pets. The NIH has renamed the National Center for Complementary and Alternative Medicine (NCCAM) in 2014 to the National Center for Complementary and Integrative Medicine (NCCIM) recognizing that Americans no longer consider "complementary" approaches as "alternatives" to conventional medical care. The NCCIM has reported that more than half of Americans report using a dietary supplement and that the scientific foundations for complementary approaches are needed.

In a recent report, pet supplements sales were reported to be \$541 billion in 2014 with a forecasted increase of 29% in 2019. Forty five percent of these sales were from pet stores, 35% from veterinarians, and the remaining 20% from on-line or major stores such as Target and Walmart. Consumers reported wanting to use supplements for joints, cardiac, skin, and digestive issues in their pets.

Supplements are defined by the AVMA as a macronutrient or micronutrient often used as a therapeutic agent. Veterinarians should understand how to use supplements safely and effectively as part of treatment plans. We consider supplements medical therapy that is integrated into conventional medical plans for our patients; therefore we prescribe supplements. Using supplements as part of treatment plans thoughtfully and effectively can improve patient outcomes and client satisfaction.

In human medicine, integrative medicine is defined as combining complementary approaches into conventional treatment plans as an integrative health care approach. The growth of integrative health care is being seen in hospitals, hospice centers, and military health facilities. The goal of integrative health care is to enhance overall health, prevent disease and alleviate debilitating symptoms in patients with complex and chronic diseases. These are the same goals we have for veterinary patients with chronic diseases. Understanding complementary therapies and integrating them into conventional veterinary treatment plans while improving patient care can increase services and revenues in private practices.

Osteoarthritis (OA) is the most common form of arthritis ranking as the 6th leading cause of disability globally in the human population. In veterinary medicine OA is the most common orthopedic disease in dogs and the prevalence of musculoskeletal disorders in all dogs has been reported as one in four at multicenter referral practices. Osteoarthritis occurs commonly in cats; however, there is a paucity of research in the role of nutrition or nutritional compounds in management. Strategies to modify the inflammatory environment present within joints can include the use of supplements.

Traditionally OA has been regarded primarily as a degenerative process and most often a consequence of aging. Treatments focused solely on symptomatic management rather than halting disease progression will fail as research revisits the role of inflammation in OA. The same inflammatory cytokines that drive rheumatoid arthritis have been shown in canine and feline OA including IL-1, IL6, and TNF α . Studies suggest IL-1 β and TNF α occupy a key role in the development of OA, with elevated levels of these cytokines and/or their gene expression reported in synovial tissues, synovial fluid and/or plasma. Investigating the role of individual supplements in

maintaining the normal anabolic and catabolic processes in the OA niche and attendant inflammatory milieu can be used as preventative, regenerative and maintenance strategies for bone or cartilage in OA.

Understanding the pathophysiology of osteoarthritis, allows the practitioner to understand how complementary supplements can be integrated into patient management plans. Specific OA supplements have been evaluated in dogs or *in vitro* using canine cells. Supplements that are evaluated include chondromodulating agents (glucosamine and/or glucosamine chondroitin sulfate), which can be prescribed parentally and orally. Other supplements that are evaluated include omega-3 (n-3) fatty acids, Boswellin or Indian frankincense, avocado/soybean unsaponifiables, and curcumin. There is evidence based research for these supplements as well as discussion of potential mode of action. Using supplements preemptively should be considered in patients at risk i.e. post orthopedic, performance/working dogs and geriatrics.

Clients are familiar with OA, perceive their pet is in pain and recognize it impacts quality of life. All too often we are euthanizing patients with poor mobility because of advanced OA. Today's owners are requesting and want plans to proactively prevent, treat and manage OA in their pets. Case examples will assist the practitioner in selecting supplements, prescribing them, and evaluating their effect in managing the patient with OA.

References available upon request

SUPPLEMENT THE ORTHOPEDIC CARE IN YOUR PRACTICE II

Donna M. Raditic DVM, DACVN

Veterinarians should consider the proactive use of supplements especially in dog breeds prone to the genetic disease, developmental orthopedic disease (DOD). Both nutrition and genetics play a role in developmental orthopedic disease (DOD) in the large breed puppy. There is evidence based research that suggests using supplements preemptively may improve outcomes in osteoarthritis. These studies and clinical experience support the proactive use of supplements in DOD breeds and other patients that may be predisposed to developing OA.

We have found owners are appreciative and responsive to using safe and prescribed supplements to prevent the development of OA. We also recommend using therapeutic joint diets as is part of a program to prevent DOD. Currently one of the therapeutic joint diets, Purina JM is labelled both for growth of puppies and maintenance of the adult dog. Case examples will enable practitioners to understand how to combine nutrition and supplements to proactively manage DOD and OA at risk patients.

Glucosamine chondroitin and n-3 fatty acids are now incorporated into therapeutic joint diets. The n-3 fatty acids currently recognized are Eicosapentaenoic acid (EPA) and Docosahexaenoic acid (DHA). It should be noted there is a reported NRC upper safe limit for n-3 fatty acids, specifically EPA. The levels currently in therapeutic joint diets allow for additional supplementation of approximately 50 mg of EPA+ DHA/kg body weight per day, but careful selection of supplement and diet is indicated. Problems with over supplementation can create dietary imbalances as well as gastrointestinal issues.

The therapeutic joint diets and their content of total n-3 fatty acids in mg per 100 kcal as well as glucosamine and chondroitin (ChSO₄) content in mg per 100 kcal of diet has been determined. Reported dosing for glucosamine supplementation is 25-50 mg/kg/day and chondroitin is 15-40mg/kg/day. Calculations demonstrate that the levels of these supplements in the therapeutic joint diets are low compared to these doses. Additional supplementation of glucosamine chondroitin can be given safely with therapeutic joint diets.

Evaluating supplements for the use in orthopedic diseases can be challenging. There are many products available on the market containing single active ingredients as well as combinations of ingredients. Selecting appropriate supplements for preventing OA in a DOD as well as the geriatric patient can be challenging. Understanding patient assessment, treatment goals, and what role nutrition and supplements play in OA will enable the astute practitioner to prescribe safe and effective joint diets and/or supplements.

It is important to note there is little regulation of supplements as compared to the pharmaceutical industry. Unlike the pharmaceutical industry and even the pet food industry, supplements are manufactured and sold with little regulation. We recommend the following as guidelines and sources of information for practitioners to consider when using supplements in the management of orthopedic patients and other diseases:

- National Animal Supplement Council (NASC) <http://www.nasc.cc/>
- Consumerlab.com
- Do you know the manufacturer?
- Does the manufacturer provide contact information that is immediately accessible?

- Are there studies on efficacy and safety?
- Products should have lot number and expiration date on the label
- Is there a guaranteed analysis?
- Can you readily identify the active ingredients? Is the recommended amount expressed in an exact measurement i.e. 1 tab is 5 grams, 1 teaspoon is 5 mls
- Can you readily identify inactive ingredients and fillers?
- Common sense, investigate, contact company
- Determine the cost by determining the cost/ active ingredient i.e. \$/mg of EPA + DHA

Recent reviews include the following:

- Vanderweerd, et al, JVIM 2012; 26:448
- Bartges J. Raditic D., in Physical Therapy & Rehabilitation 2nd edition. Millis & Levine; pg. 254-276.

References available upon request

SUPPLEMENT THE INTERNAL MEDICINE CARE IN YOUR PRACTICE

Donna M. Raditic DVM, DACVN

Veterinarians should acknowledge that owners use diets, supplements, herbs, massage, and acupuncture in their own health care and the expectation is for today's veterinarian is to have a basic knowledge of these complementary modalities for pets. The NIH arm for complementary medicine, the National Center for Complementary and Integrative Medicine (NCCIM) recognizes that Americans no longer consider complementary approaches as "alternatives" to conventional medical care. The NCCIM has reported that more than half of Americans report using a dietary supplement. Veterinarians should recognize clients will be requesting information on supplements for the healthcare and treatment of their pets.

In a large survey by MotiveQuest in 2014 veterinarians define preventative care as vaccines, neutering, and parasite control. In this survey pet owners when asked to define a preventative care program respond, "What about my pet's diet, exercise, care, play and emotional wellbeing?" This presents a significant opportunity to build strong relationships with pet owners and promote the need for preventative health care at every life stage and in disease management.

In human medicine, integrative medicine is defined as combining complementary approaches into conventional treatment plans as an integrative health care approach. The growth of integrative health care is continuing and its goals are to enhance overall health, prevent disease and alleviate debilitating symptoms in patients with complex and chronic diseases. These are the same goals we have for our pet patients with chronic internal medicine diseases. Understanding complementary therapies and integrating them into conventional veterinary treatment plans can improve patient care while increasing service and revenues in practice. In our clinical experience we have also learned that thoughtful and safe integrative therapies can play a role in preventative health care programs.

The AVMA Guidelines for Complementary and Alternative Veterinary Medicine states: "claims for safety and effectiveness ultimately should be proven by scientific method. Circumstances commonly require that veterinarian's extrapolate information when formulating a course of therapy. Veterinarians should exercise caution in such circumstances." When using supplements, we are often extrapolating, but safe supplements are routinely prescribed by veterinary internists' today especially in hepatic diseases. Other internal medicine cases could benefit from judicious supplements as part of long term management. We recognize in clinical practice that evidence based medicine may be a triad of the practitioner's clinical expertise, patient's values and expectations along with the best external evidence.

We have evaluated the evidence based research available to use safe supplements to complement conventional treatment plans for patients with urologic, gastrointestinal (GI), and dermatological diseases. These cases are seen on a daily basis by the general practitioner and can be challenging as some of these patients develop progressive symptoms and chronic disease states that require continual management. Common pathophysiology pathways are seen in these diseases to include inflammation, reactive oxidative species, barrier dysfunction and inappropriate immune responses. In our experience, using thoughtful supplements and specific nutrition concepts that are targeting these pathologic states can be integrated into conventional treatment for improved outcomes.

We will explore some of the options for using supplements and/or nutrition for improved management of internal medicine diseases. The astute practitioner will learn that the proactive use of supplements and nutrition may prevent chronic refractory disease states in their patients. Case examples will be provided to enable the practitioner to prescribe safe supplements and specific diets to improve patient outcomes.

References available upon request

SUPPLEMENT THE INTERNAL MEDICINE CARE IN YOUR PRACTICE II

Donna M. Raditic DVM, DACVN

Urology is literally the study of urine, but usually infers the study of normal processes and diseases of the lower urinary tract. In veterinary medicine, lower urinary tract disorders especially urinary tract infection (UTI) and urolithiasis occur commonly in the dog and cat. UTIs can be challenging to treat or to prevent because of their recurrent nature. Interestingly in human urology, urinary tract infections, prostatic and urolithiasis are the three most prominent lower urinary tract diseases. Strategies to prevent recurrence are similar in both human and veterinary medicine for these lower urinary tract diseases.

Urinary tract infections

It is critical for the practitioner to be able to develop rational diagnostic and therapeutic plans for the patient with a UTI. Urinary tract infection occurs when there is compromise of host defense mechanisms and a microbe adheres, multiplies and persists in the urinary tract. Normal host defenses include normal micturition, mucosal barrier, urine properties and systemic immunocompetence. Common bacterial UTI occur from ascending pathogens from the genital tract and urethra to the bladder, ureters and one or both of the kidneys. The bacterial reservoirs for a UTI are rectal, perineal, and genital bacteria, which overwhelm host defenses.

The question that is first considered is whether an infection is uncomplicated or complicated. Antibiotics are the cornerstone of treatment of bacterial UTI and ideally are selected based on culture and sensitivity. Simple infections resolve in approximately 2 weeks of therapy. Persistent or recurrent UTIs (RUTIs) that involve refractory bacterial isolates can be difficult to treat using conventional antimicrobial therapy alone. In both human and veterinary medicine, *Escherichia coli* is the most common cause of UTIs. In RUTIs, there is an increase in resistance of common urinary pathogens because of the overuse and misuse of antimicrobials.

Because of the growing concern for antimicrobial resistance, other complementary therapies are being evaluated extensively in the human literature. The evidence base use of relevant and practical complementary therapies for UTI in dogs and cats include cranberry supplements, D-mannose, oral probiotics, and herbs/herbal preparations. From evaluation of the research and clinical experience we have successfully used nutrition, D-mannose and/or probiotics in the treatment of RUTIs.

Urolithiasis

Urolithiasis occurs commonly in dogs and cats with most uroliths occurring in the lower urinary tract. It has been reported that 80 to 90% of lower urinary tract uroliths are struvite or calcium oxalate. Urolith formation is a complication of several disorders, some which are easily recognized and corrected such as infection induced struvite uroliths. Practitioners are also faced with uroliths where the underlying etiopathogenesis is unknown as seen in the formation of calcium oxalate uroliths.

Conventional therapies such as surgery and medical dissolution are the standard treatments for common urolithiasis in dogs and cats. Medical dissolution of infection induced struvite uroliths includes appropriate antibiotic therapy and feeding a struvite dissolution diet. Instead of the

conventional use of dissolution diets, the use of a urine acidifier D, L methionine 75-100mg/kg by mouth every 12 hours with appropriate antibiotic therapy has been reported for infection induced struvite dissolution.

Currently there is no known medical dissolution of calcium oxalate urolithiasis in the dog or cat; therefore, uroliths are removed surgically or by voiding urohydropropulsion. Calcium oxalate urolithiasis is recurrent and preventative strategies are warranted. The goals of dietary management recommended include reducing urine calcium and oxalate concentration, promoting high concentrations of urolith inhibitors, reducing urine acidity and promoting dilute urine.

The use of canned diets, flavoring water, adding water to dry foods, and high moisture homemade diet formulations have been used to promote dilute urine and successfully manage calcium oxalate urolithiasis. Diets higher in fiber or prebiotics may also be considered for calcium oxalate urolithiasis. Homemade diets can be used to provide a more customized approach to recurrent calcium oxalate urolithiasis and has been recommended in refractory cases. Potassium citrate is often contained in therapeutic diets designed for urolithiasis prevention, but it also can be added to a homemade diet to promote alkaline urine. A target urine pH of 6.6 to 7.5 is suggested in dogs and cats at risk of recurrence.

There has been an interest in the use of probiotics and calcium oxalate urolithiasis as mammals do not have the enzyme required to metabolize oxalate in the gut. There have been reports of reduced prevalence of *Oxalobacter formigenes*, an oxalate degrading bacterium in stone forming populations. Studies looking at the use of oxalate- degrading bacteria as probiotics have demonstrated the potential as a complementary therapy in the prevention of recurrent calcium oxalate urolithiasis.

Adverse reactions to food: dermatology and gastroenterology

An adverse reaction to food is defined as a clinically abnormal response attributed to an ingested food substance, and may be further categorized as immunologic or non-immunologic in nature. Food allergy is an immunologically mediated, reaction to ingested food. This is different than food intolerance, which is a non-immunologically mediated adverse reaction including toxic reactions, pharmacological reactions, metabolic reactions and idiosyncratic reactions.

The practitioner may easily manage a non-immunologic adverse food reaction i.e. a dog getting into the garbage, with a simple 24 hour fast and bland diet. Immunological mediated adverse food reactions such as food allergies may be more challenging. Food allergies may present alone or potentially with canine atopic disease, another difficult immune based disease.

Food allergies often include both dermatologic and gastrointestinal signs. Dermatologic signs often include pruritus, erythema, otitis and secondary pyoderma. Gastrointestinal signs may include vomiting and/or diarrhea, flatulence, perianal fistulae, frequent bowel movements and anorexia. Other potentially associated disorders which are less commonly noted include cholangiohepatitis/cholangitis, feline asthma and idiopathic epilepsy.

It should be noted that the etiopathogenesis of both food allergies and canine atopic dermatitis include the concept of disruption of the mucosal integrity and skin barrier, respectively to handle

antigenic proteins. Perhaps early recognition of a patient with recurrent dermatologic and/or gastrointestinal symptoms may alert the proactive veterinarian to intervene before the patient suffers from chronic, persistent immunologic adverse food reactions and/or atopic dermatitis. The proactive use of diet, probiotics and prebiotics as complementary therapies in at risk patients will be presented. Case examples will illustrate the use of proactive integrative therapies we have used to prevent overt and refractory disease states.

Inflammatory bowel disease

Inflammatory bowel disease (IBD) is an immune mediated disease which is **not due to food allergy and/or intolerance**. It is an idiopathic disorder that is associated with persistent or recurrent GI signs, characterized by histologic evidence of inflammation with no discernible cause. It is not due to infectious, neoplastic, or metabolic disease. We will include it in this discussion as an idiopathic immunological disease of the gut where a dysregulated immune response to components of the commensal (nonpathogenic) flora may play an important role in induction and perpetuation of chronic intestinal inflammation. There also is a genetic predisposition being identified especially in the German shepherd.

Understanding of role of mucosal barrier, antigens and the intestinal microbiome are critical to develop successful strategies to proactively manage at risk dogs and cats. Intervention with diet including therapeutic novel protein diets, hydrolyzed diets and homemade diets can be used to proactively treat the at risk patient. Probiotics and prebiotics (fiber) may also enable the astute practitioner to treat before severe disease develops and multiple immunosuppressive therapies are needed.

References available upon request

SUPPLEMENT THE CANCER CARE IN YOUR PRACTICE

Donna M. Raditic DVM, DACVN

Cancer is an important disease in dogs and represents one of the major causes of death accounting for 27% of all deaths in purebred dogs in the UK. In the absence of reliable historical tumor registries, it is difficult to know whether the prevalence of cancer in dogs is increasing; however, a number of factors may contribute to an increase in the diagnosis of cancer in dogs. As a result of improvements in health and welfare animals are living longer and cancer is generally a disease of older age. Advances in veterinary medicine, particularly diagnostics and higher expectations of the pet owning public are likely to result in an increased rate of diagnosis.

Integrative medicine is the use of complementary therapies with conventional medicine systems. “Integrative medicine” and “integrative oncology” are appropriate terms describing the adjunctive role of complementary therapies as part of multidisciplinary mainstream cancer care. These complementary therapies can include herbs, supplements, acupuncture, massage, and other therapies which are rational and evidence based. They are known to alleviate physical and emotional symptoms, improve quality of life (QOL), and may improve adherence to oncology treatment regimens. The demand for an integrative medicine approach is growing and veterinarians are being challenged to understand and provide these types of therapies.

Herbs and dietary supplements (HDS) are the most accessible form of complementary and alternative therapy. In one report 68% human population in the United States used some form of a HDS. In 2010, US herbal supplement sales exceeded \$5.2 billion. Between 67 and 87% of woman with breast cancer and those 9 years post diagnosis are reported to use supplements. These patients often do not report supplement use to their provider. In veterinary oncology, a study reported that 67% of clients used supplements with their pets. In this study, it was determined the use of complementary and alternative veterinary medicine (CAVM) was commonplace in this population.

Clients using complementary care for their own health care will want these options for their pets. The astute practitioner will embrace this and offer complementary care for cancer patients. The expectation is for today’s veterinarian is to have a basic knowledge of these alternative modalities for pets, especially with cancer, chronic illnesses and geriatrics. Because the veterinary oncology patient has a shorter lifespan and the economics of treatment(s) is different, the focus on quality of life (QOL) comes to the forefront of a treatment plan. An integrative approach to veterinary clinical oncology should target many physiological and biochemical tumor pathways while minimizing normal tissue toxicity and supporting overall well-being or QOL.

Assessment of the evidence base for the use of relevant HDS was evaluated by an extensive literature search in dog/cat or *in vitro* using dog/cat cells and then HDS in other species, including humans or *in vitro* cell lines. The relative strength of evidence has been considered, noting there are no meta-analysis and only a few randomized controlled clinical trials (RCCT) using HDS in veterinary oncology.

RCCT of herbs include the use of mushroom extracts. The use of *Coriolus versicolor* mushroom commonly referred to as cloud or turkey tail mushroom in 15 dogs with splenic hemangiosarcoma was done as a pilot study. The study enrolled 15 dogs with hemangiosarcoma

with splenectomy only and treated them with three dosing levels of the extract. The use of a known supplement containing S-adenosylmethionine (SAME) and silybin, the latter being a flavanolignan of milk thistle (*Silybum marianum*) was evaluated in an RCCT of dogs with lymphoma, mast cell tumor or histiocytic carcinoma. Dogs were prospectively randomized to receive either concurrent supplement during CCNU chemotherapy or to receive CCNU alone. The study evaluated the hepato-protective effects of the supplement when used with known hepatotoxic drugs.

There are some cancer studies in other species and many *in vitro* studies demonstrating the effects of HDS and their antitumor mechanisms. However, most of these treatments seldom progress to quality multi-institutional RCCT trials evaluating response rate and survival statistics. This is because of the nature of herbal medicine that precludes patenting; therefore funding for these types of studies is limited. Some extrapolation from these studies will be reviewed for their potential use in integrative veterinary oncology care.

References available upon request

SUPPLEMENT THE CANCER CARE IN YOUR PRACTICE II

Donna M. Raditic DVM, DACVN

Integrative medicine is the use of complementary therapies with conventional or traditional western medicine systems. “Integrative medicine” and “integrative oncology” are appropriate terms describing the adjunctive role of complementary therapies as part of multidisciplinary mainstream cancer care. These complementary therapies can include herbs, supplements, acupuncture, massage, and other therapies which are rational and evidence based. The demand for an integrative medicine approach is growing and veterinarians are being challenged to understand and provide these types of therapies.

Because the veterinary oncology patient has a shorter lifespan and the economics of treatment(s) is different, the focus on QOL comes to the forefront of a treatment plan. An integrative approach to veterinary clinical oncology using herbs, nutrition and dietary supplements can readily be used in the cancer patient to improve outcomes, overall well-being, and/or QOL.

It is well known that dietary (caloric) intake profoundly affects outcomes in cancer therapies as measured by both survival and quality of life. Practitioners can readily integrate into their oncology care the use of nutrition and supplements. All cancer patients should have a nutritional evaluation and changes made if the current diet is not optimal. We currently recommend the OA, dermatologic disease, and/or gastrointestinal therapeutic diets due to the higher digestibility of nutrients and specific nutrients (fiber, antioxidant) for oncology patients. We will also recommend and formulate complete and balanced homemade diets to be fed alone or with a specific therapeutic diet.

Specific dietary supplementations such as N-3 fatty acids, probiotics, Vitamin A, D, and E are used in cancer patients. In patients with non-regenerative anemia, we have explored the use of parental administration of B complex, B12, and iron dextrans noting improved QOL and an increase in hematocrit (HCT) and reticulocytes in oncology patients. Case examples utilizing integrative nutritional therapies and/or some complementary therapies will be presented.

The following organizations listed are an excellent source of information for veterinarians seeking more information about using an integrative approach for a cancer patient.

- American College of Veterinary Nutrition: www.acvn.org
- College of Veterinary Integrative Therapy: www.civt.edu.org
- Veterinary Information Network (message boards): www.vin.com
- International Veterinary Acupuncture Society: www.ivas.org
- American Academy of Veterinary Acupuncture: www.aava.org
- American Holistic Veterinary Medicine Association: www.ahvma.org
- Veterinary Botanical Medical Association: www.vbma.org

References available upon request